Welch Allyn CardioPerfect Workstation

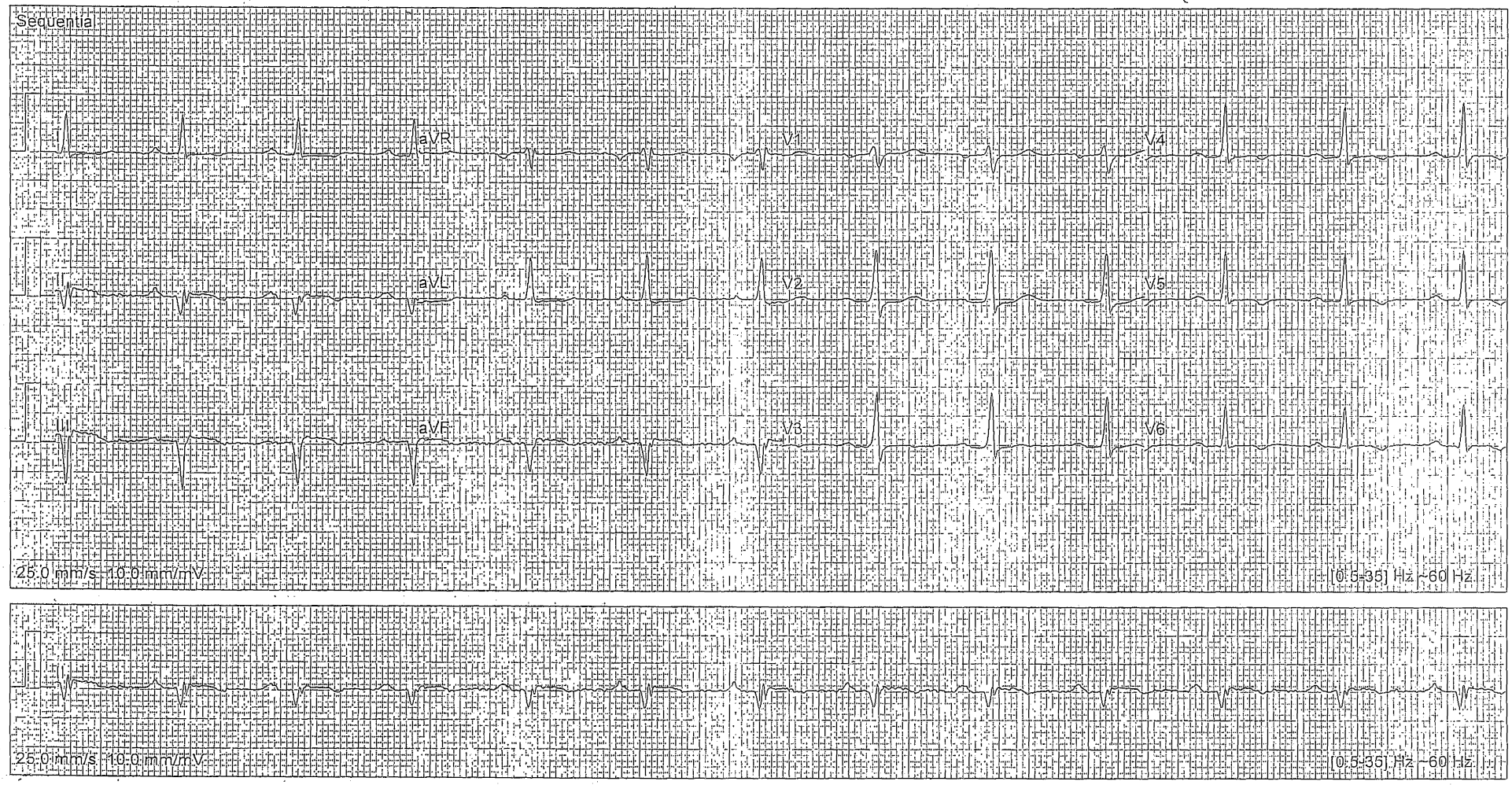
Name:	Kirk, james
Number:	119291
Gender:	Unknown
Birthdate:	1964 51 years
P / PR:	115 ms / 195 ms
QRS:	92 ms
QT / QTc / QTd:	365 ms / 396 ms / -
P/QRS/T axis:	45° / -50° / 120°
Heartrate:	78 bpm

Recorded:	7/13/2016 8:30:24 AM
Recorded by:	DAI333EKG001\docekg
Referring physician:	
Ordering physician:	
Attending physician:	
Location:	
Comment:	

UNCONFIRMED INTERPRETATION - MD SHOULD REVIEW
warning: sex not available, assumed male
sinus rhythm
inferior infarct
QS in III aVF
with Q in II
slight mid- and left-precordial repolarization disturbance, consider ischemia
small negative T in V4 V5 V6
with flat or low negative T in V3

Abnormal ECG

7/14/16



DEPARTMENT OF CORRECTIONS
Division of Adult Institutions
DOC-3021 (Rev. 8/2007)

WISCONSIN

PROGRESS NOTES

PATIENT NAME Last	First	DOC NUMBER
Kirk	James	

DATE	TIME	PROGRESS NOTES – SUBJECT, OBJECTIVE, ASSESSMENT, PLAN
6/21/16	1425	233 97.6 124/86 76 T. Helgen
		O_2 97% Peak flows 250 x 3 T. Helgen
		Stable c/o dysp — will ↑
		lisinopril to 20. f/u 2 wks
6/27/16		Patient notified via special needs committee Denied non padded chair in room. — [illegible]
7/3-16		Seen today per HSR c continued c/o of SOB, chest pain labs drawn. Has f/u c MD tomorrow. EKG was taken. In no distress today 145/85 82-16 98 98% RA. Lungs clear. Pain located on left side then Rt side, then request [illegible] for back pain. Patient able to ambulate but uses WC for distance. WC was discontinued with 231 Has f/u scheduled tomorrow. [illegible]
7/4/16	1515	228.6 97.0 124/85 87 98% T. Helgen
		In c dysp [illegible] last night. Hx of
		20+ yrs smoking. Difficult to say if
		dyspnea is cardiac vs pulm. Will get
		spirometry — ↑ lasix to 20. f/u 4 days

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B1

residual functional capacity, age, education, and work experience (20 CFR 416.912(g) and 416.960(c)).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

After careful consideration of the entire record, the undersigned makes the following findings:

1. The claimant has not engaged in substantial gainful activity since December 7, 2011, the amended alleged onset date (20 CFR 416.920(b) and 416.971 *et seq.*).

2. The claimant has the following severe impairments: coronary artery disease (CAD); cerebrovascular accident (CVA) and congestive heart failure (CHF) (20 CFR 416.920(c)).

3. The severity of the claimant's impairments medically equals a combination of the criteria of sections 11.04 and 4.02A of 20 CFR Part 404, Subpart P, Appendix 1 (20 CFR 416.920(d), 416.925, and 416.926).

In making this finding, the undersigned considered all symptoms and the extent to which these symptoms can reasonably be accepted as consistent with the objective medical evidence and other evidence, based on the requirements of 20 CFR 416.929 and SSRs 96-4p and 96-7p. The undersigned has also considered opinion evidence in accordance with the requirements of 20 CFR 416.927 and SSRs 96-2p, 96-6p and 06-3p.

The severity of the claimant's impairments medically equals a combination of listings 11.04 and 4.02A. He has a history of coronary atherosclerotic heart disease with myocardial infarction noted in 2001, 2005 and 2009. The medical evidence reflects a stent was placed in the right coronary artery in 2008. In 2009, the claimant underwent balloon angioplasty of the left circumflex artery (Exhibits 1F and 2F).

In November 2011, the claimant was admitted to the hospital with an acute CVA with significant Broca's aphasia. A history significant for CAD with previous stenting was noted. An MRI showed subacute left parietal and perisylvian cortical infarction with petechial hemorrhage likely due to recanalized vessel. He had a severely reduced ejection fraction at 38 percent. The stroke rendered him mute and upon discharge, he was referred for speech therapy to improve his language abilities as an outpatient (Exhibits 3F and 9F).

In June 2012, the claimant experienced significant chest pain and shortness of breath. He was seen in the emergency department and, given his cardiac history, admitted for further evaluation. Gated tomographic imaging revealed abnormal myocardial thickening in the lateral and inferior segments. Left ventricular ejection fraction was 17 percent. Dr. Prakash Shah, MD, opined that a defibrillator placed would be indicated (Exhibits 9F and 11F).

A follow-up echocardiogram in February 2013 was abnormal, showing significant left ventricular systolic dysfunction and ejection fraction at 38% with a global left ventricular hypokinesia (Exhibit 21F). The claimant was followed by Dr. Michel Papp, DO, who assessed NYHA Class III ischemic cardiomyopathy with decompensation with lower extremity edema.

See Next Page

C1

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The claimant was subsequently hospitalized in May 2013 with acute onset atypical chest pain. A cardiac catheterization found the previous stent of the left circumflex to be totally occluded. An echocardiogram showed inferior and posterior wall hypokinesia in the left ventricle. Systolic function was mildly to moderately reduced and the estimated ejection fraction was 35-40%. Doppler parameters were consistent with abnormal LV relaxation (grade 1 diastolic dysfunction).

Dr. Papp continues to oversee medication management. He has stressed the need for lifetime adherence to the medication regimen; however, the claimant has not always received prescribed medication during periods of incarceration (Exhibit 27F).

Independent medical examiner, Dr. Joseph Gaeta, MD, FACC, FACP, testified at the hearing. He had the opportunity to review the entire record and hear the claimant's testimony. Dr. Gaeta noted that the claimant had a long history of heart disease including three episodes of angioplasty. He also noted the history of CVA and CHF. He opined that these impairments would equal a combination of listings 11.04 and 4.02A. Dr. Gaeta further stated that this opinion would hold for the duration of the claimant's alleged disability period. The undersigned gives great weight to the independent medical expert's opinion, as it is consistent with the record when considered in its entirety.

After considering the evidence of record, the undersigned finds that the claimant's medically determinable impairments could reasonably be expected to produce the alleged symptoms and that the claimant's statements concerning the intensity, persistence and limiting effects of these symptoms are generally credible. His testimony as to symptoms and limitations is consistent with the records of treating providers.

The State agency medical consultants' physical assessments are given little weight because another medical opinion is more consistent with the record as a whole and evidence received at the hearing level shows that the claimant is more limited than determined by the State agency consultants.

4. The claimant has been under a disability as defined in the Social Security Act since December 7, 2011, the amended alleged onset date of disability (20 CFR 416.920(d)).

DECISION

Based on the application for supplemental security income filed on December 7, 2011, the claimant has been disabled under section 1614(a)(3)(A) of the Social Security Act since December 7, 2011.

The component of the Social Security Administration responsible for authorizing supplemental security income will advise the claimant regarding the nondisability requirements for these payments and, if the claimant is eligible, the amount and the months for which payment will be made.

See Next Page

C2

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/s/ Patrick J Toal

Patrick J. Toal
Administrative Law Judge

March 9, 2016
Date

C3

BLACK RIVER MEMORIAL HOSPITAL

Black River Memorial Hospital
711 West Adams Street
Black River Falls, WI 54615-
(715) 284-5361

PATIENT: KIRK, JAMES
MR #: 202200
ACCT #: 427969
DOB/Age/Sex: 1964 51 years Male

ADMISSION DATE: 7/13/2016
DISCHARGE DATE:
ATTENDING DR: Dr. Brad Martin
LOCATION: BRMH Lab

Chemistry

Other Chemistry

Collected Date: 7/13/2016
Collected Time: 09:00 CDT

Procedure		Units	Reference Range
B-type Natriuretic Peptide	38.4	pg/mL	[0.0-100.0]

LEGEND: c=Corrected, *=Abnormal, C=Critical, L=Low, H=High, f=Footnote, #=Interpretive Data, R=Ref Lab

D1

TABLE 4-4

PREVALENCE OF MYOCARDIAL INFARCTION AND UNSTABLE ANGINA AMONG SUBSETS OF PATIENTS WITH ACUTE CHEST DISCOMFORT IN THE EMERGENCY DEPARTMENT

FINDING	PREVALENCE	
	MYOCARDIAL INFARCTION, %	UNSTABLE ANGINA, %
ST elevation (≥1 mm) or Q waves on ECG not known to be old	79	12
Ischemia or strain on ECG not known to be old (ST depression ≥1 mm or ischemic T waves)	20	41
None of the preceding ECG changes but a prior history of angina or myocardial infarction (history of heart attack or nitroglycerin use)	4	51
None of the preceding ECG changes and no prior history of angina or myocardial infarction (history of heart attack or nitroglycerin use)	2	14

Note: ECG, electrocardiogram.
Source: Unpublished data from Brigham and Women's Hospital Chest Pain Study, 1997–1999.

growth factor, myeloperoxidase, and B-type natriuretic peptide (BNP); their roles are the subject of ongoing research. Single values of any of these markers do not have high sensitivity for acute myocardial infarction or for prediction of complications. Hence, decisions to discharge patients home should not be made on the basis of single negative values of these tests.

Provocative tests for coronary artery disease are not appropriate for patients with ongoing chest pain. In such patients, rest myocardial perfusion scans can be considered; a normal scan reduces the likelihood of coronary artery disease and can help avoid admission of low-risk patients to the hospital. Promising early results suggest that 64-slice computed tomography (CT) and cardiac magnetic resonance imaging (MRI) may be of sufficient accuracy for diagnosis of coronary disease that these technologies may become widely used for patients with acute chest pain in whom the diagnosis is not clear.

Clinicians frequently employ therapeutic trials with sublingual nitroglycerin or antacids or, in the stable patient seen in the office setting, a proton pump inhibitor. A common error is to assume that a response to any of these interventions clarifies the diagnosis. While such information is often helpful, the patient's response may be due to the placebo effect. Hence, myocardial ischemia should never be considered excluded solely because of a response to antacid therapy. Similarly, failure of nitroglycerin to relieve pain does not exclude the diagnosis of coronary disease.

If the patient's history or examination is consistent with aortic dissection, imaging studies to evaluate the aorta must be pursued promptly because of the high risk of catastrophic complications with this condition. Appropriate tests include a chest CT scan with contrast, MRI, or transesophageal echocardiography (TEE).

Acute pulmonary embolism should be considered in patients with respiratory symptoms, pleuritic chest pain, hemoptysis, or a history of venous thromboembolism or coagulation abnormalities. Initial tests usually include CT angiography or a lung scan, which are sometimes combined with lower extremity venous ultrasound or D-dimer testing.

If patients with acute chest discomfort show no evidence of life-threatening conditions, the clinician should then focus on serious chronic conditions with the potential to cause major complications, the most common of which is stable angina. Early use of exercise electrocardiography, stress echocardiography, or stress perfusion imaging for such patients, whether in the office or the emergency department, is now an accepted management strategy for low-risk patients. Exercise testing is not appropriate, however, for patients who (1) report pain that is believed to be ischemic occurring at rest or (2) have electrocardiographic changes not known to be old that are consistent with ischemia.

Patients with sustained chest discomfort who do not have evidence for life-threatening conditions should be evaluated for evidence of conditions likely to benefit from acute treatment (Table 4-3). Pericarditis may be suggested by the history, physical examination, and ECG (Table 4-2). Clinicians should carefully assess blood pressure patterns and consider echocardiography in such patients to detect evidence of impending pericardial tamponade. Chest x-rays can be used to evaluate the possibility of pulmonary disease.

GUIDELINES AND CRITICAL PATHWAYS FOR ACUTE CHEST DISCOMFORT

Guidelines for the initial evaluation for patients with acute chest pain have been developed by the American College

KIRK, JAMES
DOB: 10/24/1964
DOS: 07/28/2016
Attn: Tidquist, Debra
FIN: 428409
MRN: 2002200

Eau Claire Medical Clinic Oakwood
3802 W. Oakwood Mall Drive,
Eau Claire, WI, 54701
(715) 839-9280

Encounter Date: Jul 28 2016 6:57PM

Patient Information
MRN: 137751
James Kirk
PO Box 232
Black River Falls, WI 54615
DOB: 1964 (51yrs)
Gender: M
(H) (715) 284-4550

Procedure
SPIROMETRY

REQUESTING PRACTITIONER: Debra Tidquist, NP

REASON FOR TESTING: Chest pain and dyspnea.

HISTORY: The patient underwent a pre and post bronchodilator spirometry that showed an FVC of 4.71, which is 88% of predicted. The patient's FEV1 was 4.66, which is a 1% decline. The patient's FEV1 was 3.20, which is 77% of predicted with post bronchodilatory FEV1 of 3.13, which is a 2% decline. The patient's FEV1 to FVC ratio was 68%. The patient's flow volume loop was most consistent with an obstructive pulmonary defect.

IMPRESSION:
1. Mild obstructive pulmonary defect.
2. The patient did not show a significant response to bronchodilator therapy, however, this does not mean that the patient would not benefit from bronchodilators. Certainly clinical correlation is warranted.

KYLE J. DETTBARN, M.D.
EAU CLAIRE MEDICAL CLINIC

KJD/snl

cc: Debra Tidquist, NP
Black River Memorial Hospital Respiratory Therapy Department

Signatures
Electronically signed by : Kyle Dettbarn, M.D.; Aug 4 2016 10:44AM CST (Author)

F1

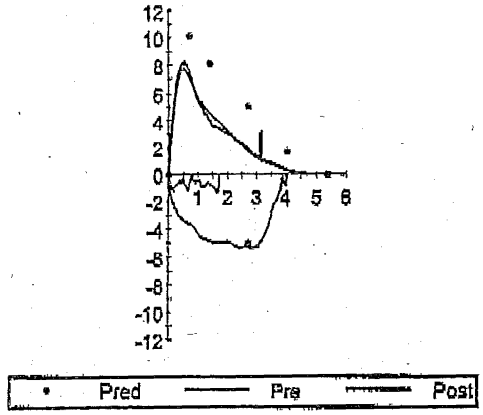
KIRK, JAMES
DOB: /1964
DOS: 07/28/2016
Attn: Tidquist, Debra
FIN: 428409
MRN: 202200

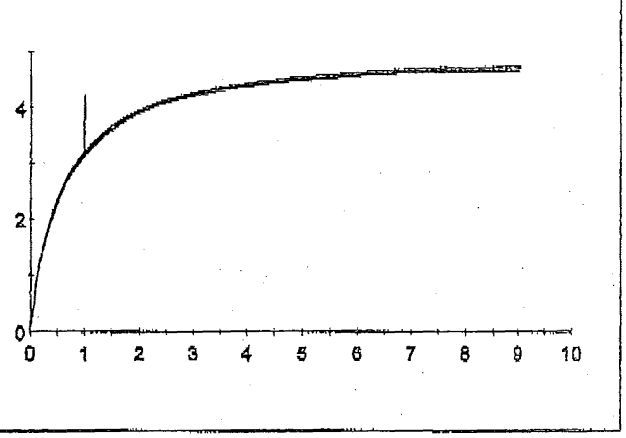
Black River Memorial Hospital
711 W Adams St
Black River Falls, WI, 54615

Name:	Kirk, James	ID:	202200	BSA:	2.24		
Tech:	Kim Schlifer, RRT	Height:	72.00	Age:	51		
Doctor:	Debra Tidquist FNP	Weight:	225.00	Sex:	Male	Race:	Caucasian

Diagnosis: Chest pain and SOB with short distances
Dyspnea: After severe exertion Cough: No Cough Wheeze: No Wheeze
Tbco Prod: Cigarette Yrs Smk: 30.0 Pks/Day: 2.0 Yrs Quit: 2.5
Medications: No respiratory meds.
Pre Test Comments: Stroke 2011, Heart Attack x3, last one was in 2009, stent placed. Tobacco use history, quit for 2.5 years. Used to work as a welder and roofer.
Post Test Comments: Patient gave good efforts. Bronchodilator used was UD Albuterol neb. 15 minute wait between pre and post maneuvers. Patient stated his breathing felt "no different" after the neb was given.

	Pre-Bronch			Post-Bronch		
	Actual	Pred	%Pred	Actual	%Pred	%Chng
--- SPIROMETRY ---						
FVC (L)	4.71	5.35	88	4.66	87	-1
FEV1 (L)	3.20	4.13	77	3.13	75	-2
FEV1/FVC (%)	68	77	88	67	87	-1
FEF 25% (L/sec)	5.14	8.23	62	4.77	57	-7
FEF 75% (L/sec)	0.90	1.74	51	0.80	46	-10
FEF 25-75% (L/sec)	2.03	3.58	56	1.88	52	-7
FEF Max (L/sec)	7.74	10.20	75	8.19	80	+5
FIVC (L)	1.73			4.05		+133
FIF Max (L/sec)	1.23			5.33		+331





REC'D AUG 08 2016

F2

Gundersen Health System
La Crosse, WI 54601
Transthoracic Echocardiographic Report
Study Date: 9/7/2016

Name: James Kirk MRN: 021110978
Provider: CHIH-SHENG J CHIANG DOB: 10/24/1964 Age: 51 years
Indication: CARDIOMYOPATHY

Conclusions:

1. Diffuse hypokinesis of the left ventricle. Inferior wall looks thinned and possibly scarred.
2. Moderately decreased LV ejection fraction.
3. Abnormal septal motion consistent with left bundle branch block.
4. Impaired relaxation pattern of LV diastolic filling.
5. Estimated filling pressures are normal.
6. No prior echo images or reports for comparison.

Description of Findings:

Study: 2D, M-mode, color Doppler and spectral Doppler.
Cardiac Rhythm: Is normal sinus rhythm.
Left Ventricle: Abnormal (paradoxical) motion consistent with left bundle branch block. Spectral Doppler shows impaired relaxation pattern of LV diastolic filling. Diffuse hypokinesis.
Left Atrium: Left atrial size is normal. Left atrial pressure is estimated to be normal.
Right Ventricle: Normal right ventricular size and function.
Right Atrium: The right atrium is normal. Right atrial pressure is estimated to be normal.
Aortic Valve: The aortic valve is normal in structure. No aortic stenosis.
Mitral Valve: The mitral valve is normal in structure. No evidence of mitral valve stenosis.
Tricuspid Valve: The tricuspid valve is normal in structure.
Pulmonic Valve: The pulmonic valve is not well visualized.
Pulmonary Artery: The pulmonary artery is not visualized.
Aorta: The ascending aorta appears normal.
Venous: Inferior vena cava is normal with normal inspiratory collapse.
Pericardium: No pericardial effusion is seen.

All Measurements and Calculations on next page.

Final

2-D and M-Mode Measurements:

	2D	M-Mode	NL Values Men	NL Value Female	Calculations	
LVIDd			4.2-5.8	3.8-5.2	LV EF% (Apex)	
LVIDs					HR	
IVSd			0.6-1.0	0.6-0.9	BSA	2.25 m²
LVPWd			0.6-1.0	0.6-0.9	LV Mass (I BSA)	
Ao Asc(d)	3.2 cm		<3.7 cm			
LA (s)			2.0-4.0 cm		Ao Asc Zscore	0.94
RV			<3.7 cm			
LA (Vol)			<20 cm			
RA (area)						

Diastology:

Mitral		NL Values	Pulmonary	NL Values	Heomdyn		NL Values
E Peak	0.3 m/s		Pulm S		Prop Vel		>50cm/s
A Peak	0.7 m/s		Pulm D		E'	0.04 m/s	
E/A	0.5	1.1-1.5	D dt		A'	0.07 m/s	
DT		160-240 ms	ar Vel	<35cm/s	E/E'	8	<12
A dur	127 msec		ar Dur				
IVRT	157 msec	50-90msec					

Aortic:

		NL Values			Severe		Severe
Vmax	0.9 m/s		AVA	3.88 cm²	<.8	AI slope	
TVI	0.12 m		Max G	3.5 mmHg	>64mmHg	AI P 1/2	<500 ms
LVOT V	0.7 m/s		Mean G	1.9 mmHg	>40mmHg	AI ERO	
LVOT TVI	0.12 m		DOI	0.75	<.25	AI Vol	
LVOT dia	2.3 cm		Planim AV		<.8	AI RF%	
						CEQ Vol	

Mitral:

	Severe		Severe
MVA	<1.0cm2	MR ERO	>40mm2
P 1/2 t	>180ms	MR Vol.	>60ml
Mean G	>10mmHg	MR radius	
MV TVI		CEQ Vol	
MV plan		MR TVI	

Tricuspid:

		NL Values		NL Values
TR Vmax	1.7 m/s		TR ERO	>40mm2
TR maxG	12.0 mmHg		TR Vol	>30ml
RAP		>5 mmHg		
RVSP		>35mmHg		
IVC insp	0.67 cm			
IVC exp	0.92 cm	<2.2 cm		

Pulmonic:

NL Values		NL Values
PV Vmax	1.0 m/s	
PV meanG		
PADP		

Height:	183 cm	HR:	bpm
Weight:	103 kg	BP:	87/55 mmHg
BSA:	2.25 m²	Gender:	M

Sonographer: MR
Site: LACROSSE
Location:INPT 3857
Imaging Limitations:
Contrast/IV documentation:

Electronically Signed By: 339176 Rajah S. Sundaram MD
9/8/2016, 1:47:41 PM

PATIENT NAME Last	First	MI	DOC NUMBER
Kirk, James			119291

DATE	TIME	PROGRESS NOTES – SUBJECT, OBJECTIVE, ASSESSMENT, PLAN
9/8/16	1100	Pt seen to F/U hosp stay – discussed c̄ pt. Discharge notes from Hosp. Pt feels that he had blockages opened however there was no blockages found c̄ angiogram. Also update pt on ~~normal~~ ERROR ECHO results + troponin results – discuss troponin levels remains normal during his stay – Reviewed c̄ pt discharge instructions + how to care for groin site + not to lift more than 10 pounds. O. Gen NAD denies chest pain or SOB today – VS stable WNL – afebrile – lungs CTA Heart S1S2 RRR ⓪MRG. Discussed c̄ pt will be F/U c̄ cardiology later this month. Pt continues to state surgeon told him they opened up his coronary arteries again reviewed procedure noted that stated no blockages were found. Pt still feels he had blockages – discussed he can discuss this c̄ next visit c̄ cardiologist. LE free from edema. Abd soft NT BS +++. ⓪hepatosplenomegaly – angio cath site – free from s/s infection some ecchymosis. – Started on ISMN 30mg ER
		A: Stable s/p angiogram & C.P today ASOB
		P: F/U c̄ me 2weeks cont POC
		Debra Tiska[illegible] APRN-BC

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H1

PATIENT NAME Last	First	MI	DOC NUMBER
Kirk,	James		119291

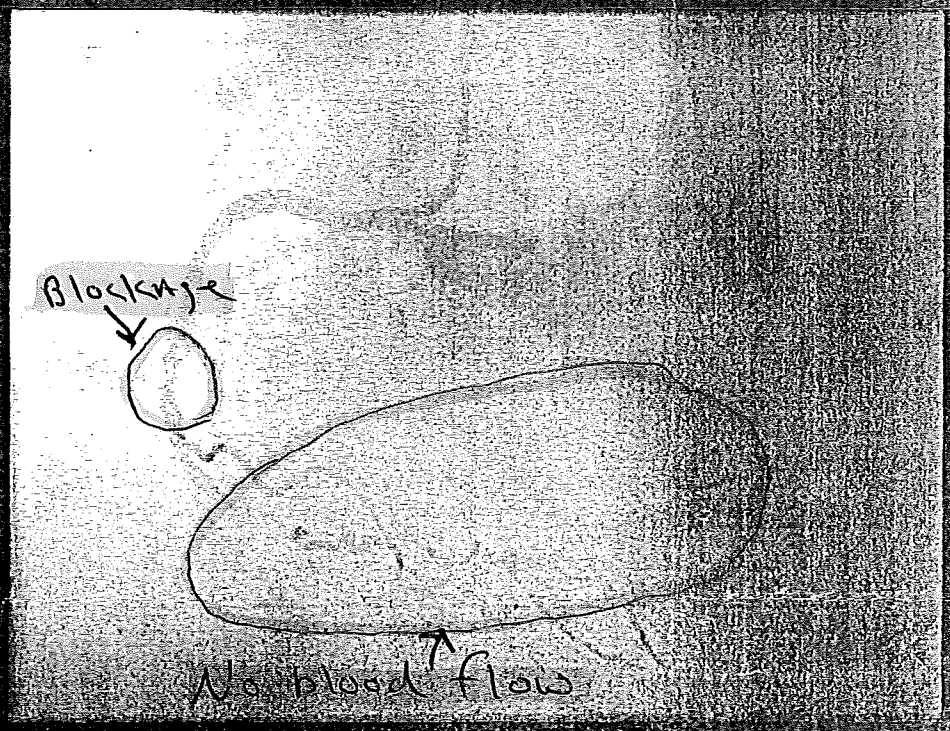
DATE	TIME	PROGRESS NOTES – SUBJECT, OBJECTIVE, ASSESSMENT, PLAN
9/1/16 1100		Seen to Flu Chest pain c/o gastric pain afraid has ulcer denies dark tarry stools or coffee ground emesis - states has pain in epigastric area. O: Gen NAD Wgt 228 BP 120/70 P 74 R 16 neck supple lungs CTAP Heart S1S2 RRR ○ MRG. labs not available - will reorder - discuss need for hemocult stools - & H. Pylori Ag - Pt agrees ☐ PPOC (started on Ranitadine 150mg bid 9/7/16) Abd soft NT ○ pain ☐ palp over epigastric area. ○ LE edema. Cath site healing well. A: ? gastric pain, BP good control chest pain good control ☐ ISMN P: H. Pylori Ag stool Cont Ranitadine Hemocult stools - Flu HTD Dec ☐ labs At end of visit reviewed ☐ PT that there was no blockages in his coronary arteries - feels stiff as learn to him - again stressed pt the Hosp notes from cardiologist continues to imply he had blockages. Again discussed ☐ pt he can review this ☐ cardiologist. Debra Toquet FNPBC

DISTRIBUTION: Original – Medical Chart, Progress Notes Section

I1

KIRK, JAMES
21110978
9/6/2016
10:05:02 AM
8 - 21/66

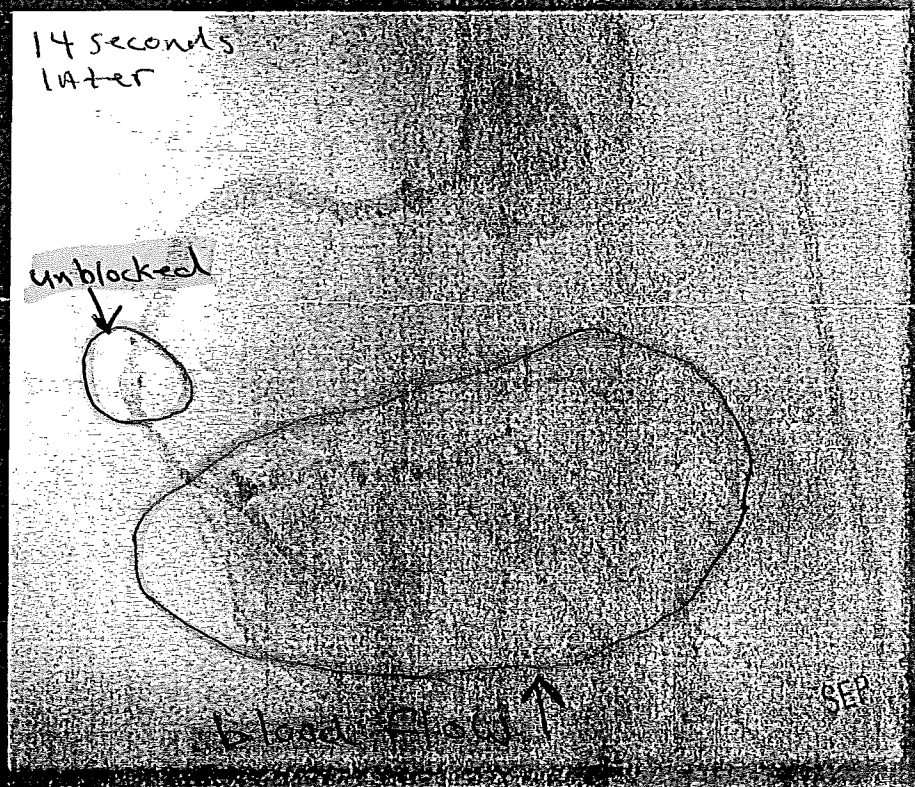
GUNDERSEN HEALTH
Perf, Subla, Mir Rauf, MD
VD11B 1509 5



30° / CRAN 1°

KIRK, JAMES
21110978
9/6/2016
10:05:16 AM
9 - 20/52

GUNDERSEN HEALTH
Perf, Subla, Mir Rauf, MD
VD11B 1509 5



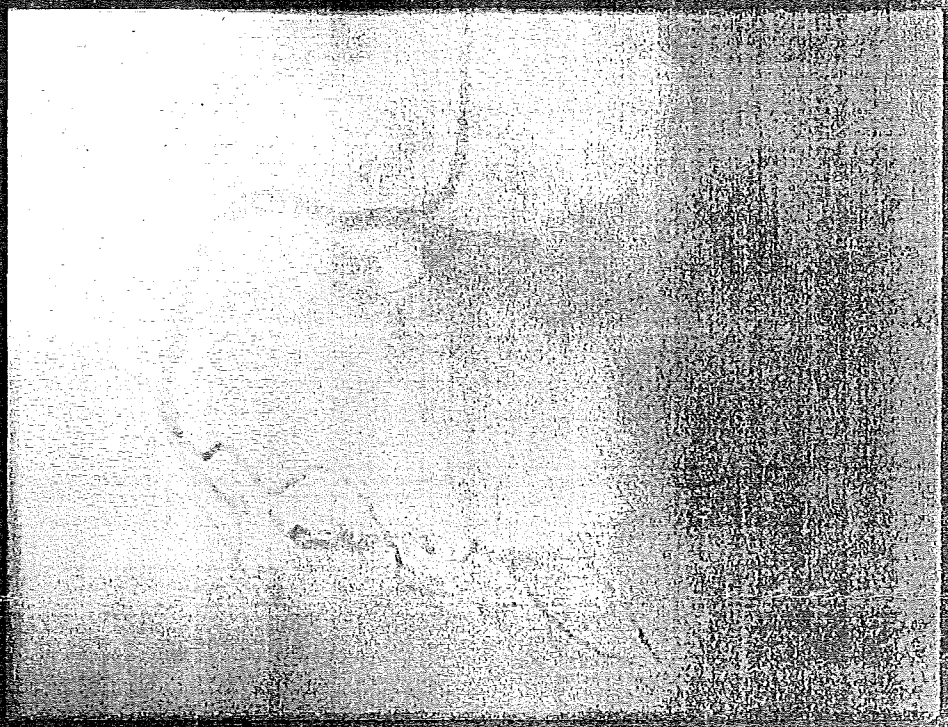
AO 1° / CRAN 27°

J1

KIRK, JAMES
21110978

9/6/2016
10:05:02 AM
8 - 21/66

GUNDERSEN HEALTH
Perf, Subtr, Mt Rauf, MD
VD11B 150915

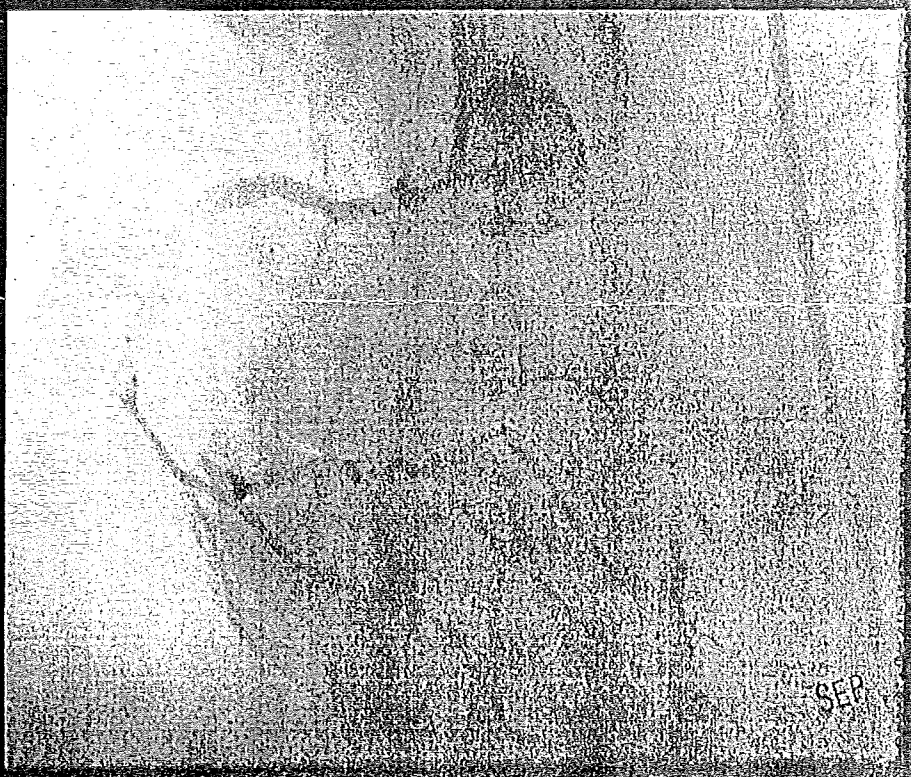


LAO 30° / CRAN 1°

KIRK, JAMES
21110978

9/6/2016
10:05:16 AM
9 - 20/52

GUNDERSEN HEALTH
Perf, Subtr, Mt Rauf, MD
VD11B 150915



LAO 1° / CRAN 27°

J2

Supplies Summary

Item Name (Model)	
Guidewire Small J 035 145 cm	Angioseal 6F
Sheath 6F Pinnacle	Pack, Surgical Cardiac Cath
Diag. RCS Catheter 6F R4	Kit, Manifold
Diag. RCS Catheter 6F R5	Kit, Transducer (Transducer)
Guiding LCS Catheter 6F CLS 3.5	Inflation Kit
Diag. RCS Catheter 6F AR1	Sheath 5F. .038 Guidewire

Medication Events

Start	Medication	Amount	Ordered by	Given by
9:49 AM	1% Lidocaine Subcut	10 ml	Sampoornima Setty MD	Sampoornima Setty MD
9:49 AM	Versed IV	1 mg	Sampoornima Setty MD	Michele Meyer RN
9:52 AM	1% Lidocaine Subcut	10 ml	Sampoornima Setty MD	Sampoornima Setty MD
9:53 AM	IV fluids IV	999 ml per hr	Sampoornima Setty MD	Michele Meyer RN
9:55 AM	Versed IV	1 mg	Sampoornima Setty MD	Michele Meyer RN
9:56 AM	Fentanyl IV	50 mcg	Sampoornima Setty MD	Michele Meyer RN
10:01 AM	IV fluids IV	100 ml per hr	Sampoornima Setty MD	Michele Meyer RN
10:05 AM	Oxygen Nasal cannula	2 l per min	Sampoornima Setty MD	Michele Meyer RN

Vital Signs

Time	SpO2 (%)	HR(BPM)	BP(mmHg)	RR (per min)	LOC
9:47 AM	98	177	84/62/67	25	
9:54 AM	94	95	131/85/104	17	2
9:58 AM	94	110	123/80/97	16	2
10:03 AM	88	88	103/71/83	11	2
10:09 AM	95	84	138/78/94	11	2
10:14 AM	99	87	124/70/93	11	2
10:19 AM	94	91	133/76/98	20	2

LOC: 0 = Not Responding; 1= Arousable On Calling; 2 = Fully Awake

Gundersen Lutheran
HEART INSTITUTE
1836 South Avenue, La Crosse, Wisconsin 54601

CARDIAC CATHETERIZATION REPORT

Patient Information			
Patient Name	KIRK, JAMES	**Date of Birth**	10/24/1964
Study Date	9/6/2016	**Age**	51 Years
MRN	21110978		

Performing Physician

Diagnostic Cardiologist	Sampoornima Setty, MD

Case Information

Indication:	STEMI	**Contrast used by ml:**	95
Site 2:	Right Femoral Vein	**Fluoro Time:**	3.7
Site 1:	Right Femoral Artery	**mGy**	693
LAB	1	**Contrast wasted by ml:**	5

Procedures

93454 - Coronary Angiography
Priority One

Cardiac Findings

RLVBR %	100	**2nd OM %**	100	**Dominance**	Right

Results Summary

I approve of all medications administered by circulating RN (s)	yes
Final Diagnosis 1. Case performed via RFA and RFV without difficulty. 2. Double vessel CAD. Proximal RCA spasm with catheter, no lesion. 3. Distal RCA after RPDA origin is chronically occluded with left to right collaterals. 4. OM2 has an occluded stent with left to left collaterals. 5. LAD is intact with only luminal irregularities. 6. Angioseal to RFA. RFV sheath is sutured to groin due to lack of peripheral IV access. Sampoornima Setty, MD FACC	
Date	9/6/2016
Time	10:20:16
Estimated Blood Loss:	Minimal
Specimen	None

Pressure Summary (mmHg)

Site	Sys	Dias	Mean
AO	128	87	101

L1

Kirk, James

MRN: 000021110978

Description: 51 year old male

H&P Date of Service: 09/06/16 1214

Chih-Sheng J Chiang, MD

zCardiology Service

Cosigned by: Michael A Witcik, MD at 09/06/16 1414

Attestation signed by Michael A Witcik, MD at 09/06/16 1414

I have seen and examined the patient and reviewed the resident's findings, assessment, and plan as written. My participation and any additional findings and comments on the assessment and plan are as follows.

Assessment/Plan:

51 y/o w/PMH of known CAD transferred as P1 due to chest pain and new LBBB. Coronary angiography with patent LAD, CTO of OM and dRCA with collaterals. Pain now resolved, hemodynamically/electrically stable. No indication for PCI/CABG.
- Continue CAD meds titrate as able.
- Update TTE for structure/function.
- Serial biomarkers/EKGs.

Total time spent on case today: 30 minutes and at least 50% of time was spent on patients unit coordinating patient's care and/or counseling patient.

Mr. Kirk's current Medical Level of Care is **observation.**
OBSERVATION level of care is appropriate because I am NOT certain that hospital confinement (including time spent in the ED or at an outside institution prior to transfer) will be medically necessary for at least 2 midnights.

Michael A. Witcik, M.D.
Department of Cardiology

CARDIOLOGY HISTORY & PHYSICAL - Attending is Dr. Witcik

9/6/2016 James Kirk 000021110978

MISSION LOCATION: GUNDERSEN HEALTH SYSTEM

M1

CHIEF COMPLAINT: Chest pain at rest

HISTORY OF PRESENT ILLNESS:
James Kirk is a 51 y.o. Caucasian male who presents with a 9-week history of intermittent chest discomfort that suddenly worsened last night while in the bathroom. He has had a history of repeat MI starting in 2005 s/p stent placement in OM2 and subsequent angioplasty as well as a history of CVA and heart failure with decreased EF (patient reports 17%). Over these last few weeks he has also experienced increased shortness of breath and waxing/waning right leg swelling that disappears with rest. He has gone to the Health Service Unit several times to report his complaints but it was decided that his chest discomfort was the result of COPD and no coronary intervention was necessary.

Last night around 8 PM he was in the bathroom getting ready for bed when he felt stabbing chest pain rated 9/10. The pain felt radiated to his left shoulder and left jaw. He became lightheaded and fell but did not lose consciousness. He was taken to the ED at Black River Falls by ambulance when he was found. His troponin was not elevated and EKG did not show an ST elevation. He says an IV line was not placed because of his history of IV drug abuse. No nitroglycerin was given either. He did receive a fentanyl patch, which resolved his chest pain. At admission in Gundersen Lacrosse his chest discomfort was rated a 1/10 without pain medication or nitroglycerin drip.

Past Medical History
1. Echocardiogram obtained 11/16/2015, revealed mildly dilated left ventricle with mildly reduced global systolic function, with ejection fraction estimated to be 40%. There was an akinetic inferolateral segment. Diastolic dysfunction was noted. Mild left atrial enlargement was seen. No gross valvular abnormality was discerned.
2. CVA in 2011
3. History of pneumonia.
4. History of back pain.
5. History of hepatitis C.
6. Status post left thumb surgery.
7. Status post the index finger and 3rd finger of his left hand with they "tips amputated" in an industrial accident.
8. Status post bilateral knee surgery.
9. Status post left shoulder rotator cuff repair.

Home Medications
1. Ranitidine 150 milligrams by mouth twice a day.
2. Carvedilol 3.125 mg by mouth twice a day.
3. Triamterene-hydrochlorothiazide 37.5 milligrams/25 milligrams 1 tablet by mouth daily.
4. Naproxen 1 tablet by mouth twice a day.
5. Selenium as directed.
6. Aspirin 325 milligrams by mouth daily.
7. Lisinopril 5 milligrams by mouth daily.
8. Simvastatin 40 milligrams by mouth nightly.

HABITS:
The patient quit smoking 2 years ago when he was incarcerated. 40+ smoking year history. Drinks 2 cups of caffeinated coffee a day.

SOCIAL HISTORY:

M2

Patient has a long-standing girlfriend (they have been together for 26 years). He used to work as a roofer and welder.

FAMILY HISTORY:
Patient's father is age 77, and alive and well. Patient's mother is alive and well, and he has one sister who is alive and well, one brother who is alive and well. Two sons who are alive and well. One daughter who is alive and well.

Primary Care Providers:
NEED TO EST PCP, MD

Resuscitation Status: Full.

REVIEW OF SYSTEMS:
A complete review of systems was negative except for that mentioned in the history of present illness

PHYSICAL EXAMINATION:

Patient Vitals for the past 12 hrs:

	BP	Temp	Heart Rate (monitor)	Resp	SpO2	Flow (L/min)
09/06/16 1040	172/67	36.9 °C (98.5 °F)	91	30	(!) 87 %	-
09/06/16 1044	-	-	-	-	-	2
09/06/16 1045	109/68	-	90	22	92 %	-
09/06/16 1046	-	-	90	-	-	-
09/06/16 1100	124/78	-	90	27	92 %	-
09/06/16 1115	118/80	-	90	17	93 %	-
09/06/16 1130	117/80	-	86	22	93 %	-
09/06/16 1145	-	-	91	-	93 %	-
09/06/16 1200	-	-	87	-	93 %	-
09/06/16 1300	120/76	-	90	39	94 %	-
09/06/16 1315	61/46	-	101	36	94 %	-
09/06/16 1330	103/66	-	93	16	93 %	-
09/06/16 1345	106/67	-	100	35	95 %	-
09/06/16 1400	143/93	-	101	-	96 %	-

Intake/Output Summary (Last 24 hours) at 09/06/16 1409
Last data filed at 09/06/16 1405

	Gross per 24 hour
Intake	240 ml
Output	0 ml
Net	240 ml

General: Patient appears comfortable and in no acute distress.
HEENT: Head is atraumatic, normocephalic. Pupils are equal, round and reactive to light. No scleral icterus. Conjunctiva are pink and without injection. Midline uvula. Dentition intact.
Neck:

Neck is supple with no carotid bruits to auscultation.Thyroid is not palpable.

Lymphatic: There is no cervical adenopathy or tenderness to palpation.

Respiratory: Lungs are clear to auscultation and percussion bilaterally.

Cardiovascular: Regular rate and rhythm. Normal S1 and S2. No murmurs, rubs, or gallops. Radial and dorsalis pedis and are 2+ bilaterally. No jugular venous distention present. No pretibial edema noted.

Abdomen: Normal to visual inspection. Normoactive bowel sounds. Non-tender to palpation. No masses are appreciated.

Skin: No skin rashes or lesions to inspection or palpation.

Neurologic: Higher functions are within normal limits. Minor speech defect attributed to CVA from 2013. Cranial nerves II through XII are grossly intact and symmetric. Sensation is intact to light touch in the upper and lower extremities bilaterally. Reflexes: 2+ and symmetric bilaterally. Coordination and Gait: Within Normal limits.

SKIN INTACT? Groin access site bandaged from left heart catheterization.

CATHETER/LINES ON ADMISSION: Groin access kept in due to difficulty in maintaining access

INVESTIGATIONS:

Recent Results (from the past 20 hour(s))

LAB TROPONIN T (Now, if not already done)

Result	Value	Ref Range
TROPONIN T	<0.01	<0.03 ng/ml

LAB CREATININE

Result	Value	Ref Range
CREATININE	0.66 (L)	0.70 - 1.30 mg/dL
ESTIMATED GFR	112	mL/min/1.73m2

Date of ECHO: Pending.

Cath date: 9/6/16

1. Case performed via RFA and RFV without difficulty.
2. Double vessel CAD. Proximal RCA spasm with catheter, no lesion.
3. Distal RCA after RPDA origin is chronically occluded with left to right collaterals.
4. OM2 has an occluded stent with left to left collaterals.
5. LAD is intact with only luminal irregularities.
6. Angioseal to RFA. RFV sheath is sutured to groin due to lack of peripheral IV access.

MEDICAL DECISION MAKING:

I have personally independently reviewed and interpreted lab results and EKG
In addition, I have discussed the test results with the Attending physician.

MH

Mr. Kirk's current Medical Level of Care is Observation

OBSERVATION level of care is appropriate because I am NOT certain that hospital confinement (including time spent in the ED or at an outside institution prior to transfer) will be medically necessary for at least 2 midnights.

ASSESSMENT & PLAN:

Active Hospital Problems

Diagnosis	POA
• Unstable angina (*)	Unknown
• Chronic heart failure (*)	Unknown

Stable Angina - negative troponin, no ST elevation on EKG
- continue chewable aspirin 81 mg po qday
- start atorvastatin 80 mg po qday
- continue lisinopril 20 mg po qday
- left heart catheterization showed stable occlusion of OM2 stent and distal RCA with collateralization, indicating long-standing process. Negative troponin 12 hours after chest pain and lack of ST elevation on EKG on admission supports that no acute changes has occurred.
- Echo on 9/6 PM

Heart Failure with reduced Ejection Fraction
- continue carvedilol 12.5 mg po BID
- continue lasix 20 mg po BID

DVT prophylaxis: ambulation
GI prophylaxis: ranitidine 150 mg po BID
Diet: General diet

Dispo: Likely discharge tomorrow

Chih-Sheng J Chiang, MD
Transitional Year Resident
Pager: 0850

Admission (Discharged) on 9/6/2016

M5

Kirk, James

MRN: 000021110978

Description: 51 year old male

Progress Notes Date of Service: 09/07/16 0852

Chih-Sheng J Chiang, MD

zCardiology Service

Cosigned by: Michael A Witcik, MD at 09/07/16 1312

Attestation signed by Michael A Witcik, MD at 09/07/16 1312

I have seen and examined the patient and reviewed the resident's findings, assessment, and plan as written. My participation and any additional findings and comments on the assessment and plan are as follows.

No issues overnight. VSS. Serial biomarkers negative.

Assessment/Plan:

51 y/o w/PMH of known CAD transferred as P1 due to chest pain and new LBBB. Coronary angiography with patent LAD, CTO of OM and dRCA with collaterals. Pain now resolved, hemodynamically/electrically stable. No indication for PCI/CABG. Findings not c/w myocardial necrosis.
- Continue CAD meds titrate as able. Trial of imdur.
- Update TTE for structure/function.
- Anticipate d/c today.

Total time spent on case today: 25 minutes and at least 50% of time was spent on patients unit coordinating patient's care and/or counseling patient.

Michael A. Witcik, M.D.
Department of Cardiology

CARDIOLOGY PROGRESS NOTE - Attending is Dr. Witcik

9/7/2016 James Kirk 000021110978

Admit Date: 9/6/2016 Hospital Days: 1

ASSESSMENT & PLAN

Active Hospital Problems

Diagnosis POA

- Unstable angina (*) Unknown
- Chronic heart failure (*) Unknown

1. Stable Angina - negative troponin, no ST elevation on EKG, stable LBBB
- left heart catheterization from 9/6 AM showed stable occlusion of OM2 stent and distal RCA with collateralization, indicating long-standing process. Negative troponin 12 hours after chest pain and lack of ST elevation on EKG on admission supports that no acute changes has occurred.
- continue aspirin 81 mg po qday
- start atorvastatin 80 mg po qday
- continue lisinopril 20 mg po qday - watch BP
- start Imdur 30 mg po qday for ongoing chest discomfort
- Echo planned for 9/7 AM

2. Heart Failure with reduced Ejection Fraction
- continue carvedilol 12.5 mg po BID
- continue lasix 20 mg po BID

DVT prophylaxis: heparin 5000 units TID
GI prophylaxis: ranitidine 150 mg po BID
Diet: General diet

Dispo: Discharge in PM after echo

SUBJECTIVE:
James Kirk has done well overnight. Had chest discomfort last night but was minimal compared to previous night. Denied any shoulder pain, back pain, leg swelling or shortness of breath.

OBJECTIVE:
Vital Signs Last 24 Hours:
Temp: [36.4 °C (97.5 °F)-36.9 °C (98.5 °F)] 36.4 °C (97.5 °F)
BP: (61-172)/(46-93) 92/61
Heart Rate (monitor): [70-106] 75
Resp Rate: [10-42] 21
O2 Sat (%): [87 %-97 %] 94 %
Flow (L/min): [2] 2
Rhythm: normal sinus with stable LBBB
DAILY Weight: 102.7 kg (226 lb 6.6 oz)
I/O data for the last 2 completed shifts (starting at 4am/4pm):
In: 620 [P.O.:620]
Out: 225 [Urine:225]

General: alert, oriented. No acute distress.
HEENT: mucous membranes moist, no enlarged lymph nodes.
Respiratory: clear to auscultation B/L, trachea midline, no subcutaneous air
Cardiovascular: Regular rate, S1, S2 normal, no murmur, rub, or gallop, extremities are warm and well perfused, no LE edema.
Abdomen: soft, non-tender. Bowel sounds positive. No masses, no organomegaly.
Neurologic: alert, no gross FNDs, Moves all four extremeties equally.

DISCHARGE PLANNING SECTION:
Destination: Jail
Durable Medical Equipment: None

TESTS PENDING AT DISCHARGE: None

SUMMARY OF ADMISSION CHIEF COMPLAINT, HISTORY OF PRESENT ILLNESS, PERTINENT EXAM, LAB & RADIOLOGY STUDIES:
James Kirk is a 51 y.o. Caucasian male who presents with a 9-week history of intermittent chest discomfort that suddenly worsened last night while in the bathroom. He has had a history of repeat MI starting in 2005 s/p stent placement in OM2 and subsequent angioplasty as well as a history of CVA and heart failure with decreased EF (patient reports 17%). Over these last few weeks he has also experienced increased shortness of breath and waxing/waning right leg swelling that disappears with rest. He has gone to the Health Service Unit several times to report his complaints but it was decided that his chest discomfort was the result of COPD and no coronary intervention was necessary.

On 9/5 around 8 PM he was in the bathroom getting ready for bed when he felt stabbing chest pain rated 9/10. The pain felt radiated to his left shoulder and left jaw. He became lightheaded and fell but did not lose consciousness. He was taken to the ED at Black River Falls by ambulance when he was found. His troponin was not elevated but EKG showed a new LBBB. He says an IV line was not placed because of his history of IV drug abuse. No nitroglycerin was given either. He did receive a fentanyl patch, which resolved his chest pain. At admission in Gundersen Lacrosse his chest discomfort was rated a 1/10 without pain medication or nitroglycerin drip.

HOSPITAL COURSE: Coronary angiography showed patent LAD, CTO of OM and dRCA with collaterals. No intervention was performed as no acute disease process was identified. He continued to have some 1/10 chest discomfort overnight but nothing like he experienced the prior night. He was started on a trial of Imdur, which relieved his chest pain completely. Echo showed LVEF ~40% with no significant change in wall motion abnormalities compared to previous echo. He has remained hemodynamically and electrically stable since admission and his troponin has been negative since admission.

He will be receiving his 1-week follow up with with his prison PCP and his 1 month cardiology follow up in Gundersen Lacrosse with an NP in Cardiology.

Greater than 30 minutes spent on discharge? Yes

Chih-Sheng J Chiang, MD

Admission (Discharged) on 9/6/2016

N3

BLACK RIVER MEMORIAL HOSPITAL
Excellence Always

Black River Memorial Hospital
711 West Adams Street
Black River Falls, WI 54615-
(715) 284-5361

PATIENT:	KIRK, JAMES	ADMISSION DATE:	5/12/2017
MR #:	202200	DISCHARGE DATE:	
ACCT #:	445539	ATTENDING DR:	Antonelli,Darrin MD
DOB/Age/Sex:	1964 52 years Male	LOCATION:	BRMH Spec Serv

History and Physical

Document Name:	History and Physical
Service Date/Time:	5/12/2017 09:33 CDT
Result Status:	Transcribed
Perform Information:	Millis,Dena as proxy for Antonelli,Darrin MD (5/12/2017 10:45 CDT)
Sign Information:	

DATE OF SERVICE: 05/12/2017

Please see consultation for details. Mr. Kirk returns after a followup and workup regarding right upper quadrant pain. The patient had an echocardiogram repeated. I reviewed the records at Gundersen Lutheran Clinic. The patient was seen by the cardiologist. The patient has had multiple angioplasties and stents performed. The last echocardiogram showed a dismal ejection fraction of 28%. This most recent echocardiogram shows improvement in his overall cardiac function with an LV function ejection fraction of 68%. In the meantime to address the patient's right upper quadrant pain, he had a nuclear med HIDA scan that shows a reduced ejection fraction at 26%. This corroborates the gallbladder inflammation seen on this ultrasound. The patient represents. I have re-questioned his pain. The patient has some low-grade chronic right upper quadrant pain affected by position. The patient denies any other symptoms.

PAST MEDICAL HISTORY:

1) Congestive heart failure.
2) Low back pain.
3) Hypertension.
4) GERD.
5) Hypercholesterolemia.
6) Hepatitis C.

PAST SURGICAL HISTORY:

1) Bilateral knee surgery.
2) Left thumb reconstruction.
3) Left shoulder surgery.

Black River Memorial Hospital

Patient:	KIRK, JAMES	Admit:	5/12/2017
MRN:	202200	Discharge:	
FIN:	445539	Admitting:	Antonelli,Darrin MD
DOB/Age/Sex:	10/24/1964 52 years Male		

History and Physical

MEDICATIONS:

1) Naproxen.
2) Isosorbide.
3) Ranitidine.
4) Nitroglycerin.
5) Lisinopril.
6) Furosemide.
7) Carvedilol.
8) Atorvastatin.
9) Aspirin.

ALLERGIES: Tetanus toxoid.

REVIEW OF SYSTEMS: Review of the following systems including constitutional, ear, eye, nose, mouth and throat; cardiovascular; gastrointestinal; respiratory; musculoskeletal; genitourinary; neurological; integumentary; endocrine; psychiatric; hematologic and lymphatic all negative except as noted above.

PHYSICAL EXAMINATION:
GENERAL: In general this is a 52-year-old male in no acute distress, alert, awake and oriented to time, place and person.
HEENT – Normocephalic, atraumatic. PERRLA, EOMI.
NECK: Supple, no JVD, no carotid bruits.
LUNGS: Lungs clear to auscultation, no rales, rhonchi, or wheezes.
HEART: Regular rate and rhythm, no murmur detected.
ABDOMEN: Soft, nontender, nondistended, bowel sounds normal, active. No midline pulsatile masses. Negative Murphy's sign. No masses.
MUSCULOSKELETAL: No gait abnormalities, good range of motion all four extremities, no motor deficits.
NEURO: Neuromuscular, cranial nerves intact, no focal deficits.

Laboratory studies pending.

ASSESSMENT/PLAN: Biliary dyskinesia. The nature of this disease, the risks, benefits, alternatives and complications regarding surgery were discussed. The patient will consent to a laparoscopic cholecystectomy. Risks including but not limited to bleeding, infection, injury to bowel, bladder and blood vessel was discussed.

02

Black River Memorial Hospital

Patient:	KIRK, JAMES	Admit:	5/12/2017
MRN:	202200	Discharge:	
FIN:	445539	Admitting:	Antonelli,Darrin MD
DOB/Age/Sex:	10/24/1964 52 years Male		

History and Physical

The possibility of bile duct injury with the need for specialty repair was discussed. The possibility of open surgery was discussed as well. The patient will consent to a laparoscopic cholecystectomy. I would recommend a preoperative history and physical be performed at JCI. This should include preoperative laboratory studies including a CBC, kidney panel, liver function test and PT/INR. I would also request that a copy of the patient's ultrasound be sent with his paperwork for his operation as I would like to review that ultrasound. The patient will be scheduled as soon as possible.

CC: DEB TIDQUIST, APRN-BC

Antonelli, Darrin MD

[Transcribed on: 05/12/2017 10:45 CDT]
DM

Clinical Documents

Document Name:	History and Physical
Service Date/Time:	5/12/2017 09:33 CDT
Result Status:	Transcribed
Perform Information:	Millis,Dena as proxy for Antonelli,Darrin MD (5/12/2017 10:45 CDT)
Sign Information:	

DATE OF SERVICE: 05/12/2017

Please see consultation for details. Mr. Kirk returns after a followup and workup regarding right upper quadrant pain. The patient had an echocardiogram repeated. I reviewed the records at Gundersen Lutheran Clinic. The patient was seen by the cardiologist. The patient has had multiple angioplasties and stents performed. The last echocardiogram showed a dismal ejection fraction of 28%. This most recent echocardiogram shows improvement in his overall cardiac function with an LV function ejection fraction of 68%. In the meantime to address the patient's right upper quadrant pain, he had a nuclear med HIDA scan that shows a reduced ejection fraction at 26%. This corroborates the gallbladder inflammation seen on this ultrasound. The patient represents. I have re-questioned his pain. The patient has some low-grade chronic right upper quadrant pain affected by position. The patient denies any other symptoms.

PAST MEDICAL HISTORY:

Black River Memorial Hospital

Patient: KIRK, JAMES
MRN: 202200
FIN: 445539
DOB/Age/Sex: /1964 52 years Male

Admit: 5/12/2017
Discharge:
Admitting: Antonelli,Darrin MD

Clinical Documents

1) Congestive heart failure.
2) Low back pain.
3) Hypertension.
4) GERD.
5) Hypercholesterolemia.
6) Hepatitis C.

PAST SURGICAL HISTORY:

1) Bilateral knee surgery.
2) Left thumb reconstruction.
3) Left shoulder surgery.

MEDICATIONS:

1) Naproxen.
2) Isosorbide.
3) Ranitidine.
4) Nitroglycerin.
5) Lisinopril.
6) Furosemide.
7) Carvedilol.
8) Atorvastatin.
9) Aspirin.

ALLERGIES: Tetanus toxoid.

REVIEW OF SYSTEMS: Review of the following systems including constitutional, ear, eye, nose, mouth and throat; cardiovascular; gastrointestinal; respiratory; musculoskeletal; genitourinary; neurological; integumentary; endocrine; psychiatric; hematologic and lymphatic all negative except as noted above.

04

Black River Memorial Hospital

Patient: KIRK, JAMES
MRN: 202200
FIN: 445539
DOB/Age/Sex: 10/24/1964 52 years Male

Admit: 5/12/2017
Discharge:
Admitting: Antonelli,Darrin MD

Clinical Documents

PHYSICAL EXAMINATION:
GENERAL: In general this is a 52-year-old male in no acute distress, alert, awake and oriented to time, place and person.
HEENT – Normocephalic, atraumatic. PERRLA, EOMI.
NECK: Supple, no JVD, no carotid bruits.
LUNGS: Lungs clear to auscultation, no rales, rhonchi, or wheezes.
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MUSCULOSKELETAL: No gait abnormalities, good range of motion all four extremities, no motor deficits.
NEURO: Neuromuscular, cranial nerves intact, no focal deficits.

Laboratory studies pending.

ASSESSMENT/PLAN: Biliary dyskinesia. The nature of this disease, the risks, benefits, alternatives and complications regarding surgery were discussed. The patient will consent to a laparoscopic cholecystectomy. Risks including but not limited to bleeding, infection, injury to bowel, bladder and blood vessel was discussed. The possibility of bile duct injury with the need for specialty repair was discussed. The possibility of open surgery was discussed as well. The patient will consent to a laparoscopic cholecystectomy. I would recommend a preoperative history and physical be performed at JCI. This should include preoperative laboratory studies including a CBC, kidney panel, liver function test and PT/INR. I would also request that a copy of the patient's ultrasound be sent with his paperwork for his operation as I would like to review that ultrasound. The patient will be scheduled as soon as possible.

CC: DEB TIDQUIST, APRN-BC

Antonelli, Darrin MD

[Transcribed on: 05/12/2017 10:45 CDT]
DM

REC'D MAY 12 2017

05

MOBILE ULTRASOUND SERVICES

American Diagnostic Services
[redacted]
Pennsauken, NJ [redacted]
800.837.8496

ECHOCARDIOGRAM REPORT

This report is based solely upon the echocardiogram examination. Correlation with the clinical examination is essentail.

Facility : JACKSON CORRECTIONAL - 22232
Patient Name : Kirk, James MRN/File # : 119291 DOB : 10/24/1964 Gender : M
Claim Number : 20343513 Order / Accession Number :
Ordering Physician : DEBRA L TIDQUIST APRNBC
Reporting Physician : ELIZABETH KLODAS MD Sonographer : BRITTANY WEEDE
Date of Service : 11/16/2015 Report Date : 11/19/2015 Report Time : 06:27 AM

ECHOCARDIOGRAM:

CLINICAL INDICATIONS: Cerebrovascular accident.

Height: 6 0".
Weight: 220 lbs.
Blood Pressure: Not available.

RECD NOV 19 2015

MEASUREMENTS: Diastolic LV Dimension: 5.9 cm. (Normal 3.6-5.6 cm)
Systolic LV Dimension: 4.7 cm. (Normal 2.3-3.9 cm)
Interventricular Septum: 1.1 cm. (Normal 0.6-1.1 cm)
Posterior Wall: 1.1 cm. (Normal 0.6-1.1 cm)
Left Atrial Dimension: 4.0 cm. (Normal 1.9-4.0 cm)
Aortic Root Dimension: 3.5 cm. (Normal 2.0-3.7 cm)

FINDINGS:
1. Trans-thoracic echocardiography is performed in 2 dimensional, M-Mode, Doppler and color flow. Image quality was technically difficult secondary to patient imaging characteristics.

IMPRESSIONS:
1. Mildly dilated left ventricle with mildly to moderately reduced global systolic function. Estimated ejection fraction 40%.
2. Akinetic inferior and inferolateral segments at mid ventricle and base, consistent with previous ischemic injury.
3. Diastolic relaxation abnormality of left ventricle.
4. Mild left atrial dilatation.
5. Poorly visualized right heart chambers.
6. Incompletely visualized aortic valve. No detected aortic stenosis or insufficiency.
7. Functionally unremarkable mitral, tricuspid and pulmonic valves.
8. No masses, thrombi shunts or pericardial effusions identified with limitations

11-20-15 p

P1

MOBILE ULTRASOUND SERVICES

American Diagnostic Services
6581 N. Park Drive, Suite 6B
Pennsauken, NJ 08109
800.837.8496

ECHOCARDIOGRAM REPORT

This report is based solely upon the echocardiogram examination. Correlation with the clinical examination is essentail.

Facility : JACKSON CORRECTIONAL - 22232

Patient Name : Kirk, James MRN/File # : 119291 DOB : 10/24/1964 Gender : M

Claim Number : 20343513 Order / Accession Number :

Ordering Physician : DEBRA L TIDQUIST APRNBC

Reporting Physician : ELIZABETH KLODAS MD Sonographer : BRITTANY WEEDE

Date of Service : 11/16/2015 Report Date : 11/19/2015 Report Time : 06:27 AM

of the study.
9. Sinus rhythm incidentally noted during echocardiographic imaging.

Electronically Signed By ELIZABETH KLODAS, MD

401298/677993689/MH
DT: 11/19/2015 07:17
DD: 11/19/2015 05:22

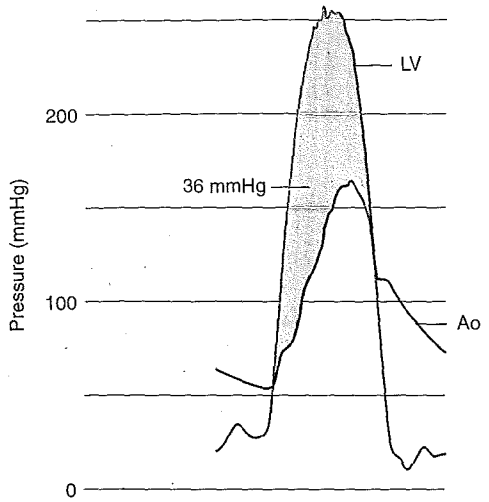

FIGURE 13-4

Severe aortic stenosis. Hemodynamic findings show the left ventricular (LV), and aortic (Ao) pressure with a slow systolic rise and large (96 mmHg) systolic gradient (shaded area) corresponding to critical aortic stenosis (aortic valve area 0.5 cm^2). *[From BA Carabello, W Grossman, in Grossman's Cardiac Catheterization, Angiography, and Intervention, 7th ed, DS Baim (ed). Baltimore, Lippincott Williams & Wilkins, 2006.]*

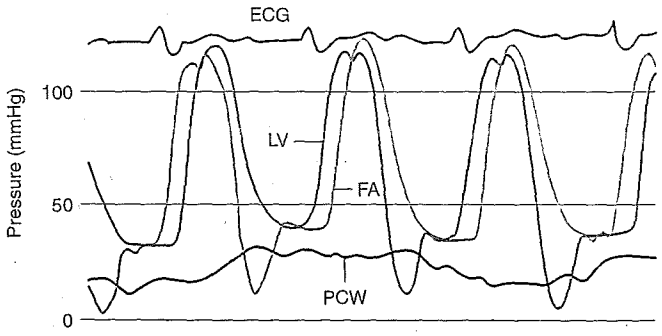

FIGURE 13-5

Severe aortic regurgitation. There is equilibration between the left ventricular (LV) and aortic or femoral artery (FA) pressures in diastole. Also, LV diastolic pressure exceeds pulmonary capillary wedge (PCW) pressure early in diastole, indicating premature closure of the mitral valve (a characteristic feature of severe aortic regurgitation). ECG, electrocardiogram. *[From W Grossman, in Grossman's Cardiac Catheterization, Angiography, and Intervention, 7th ed; DS Baim, W Grossman (eds). Baltimore, Lippincott Williams & Wilkins, 2006.]*

intimal flap separating the true and false aortic lumina (Chap. 38). Aortography may also be used to identify the origin of patent saphenous vein grafts which cannot be cannulated selectively and have not been identified by the cardiac surgeon by placement of a radiopaque marker at the graft origin.

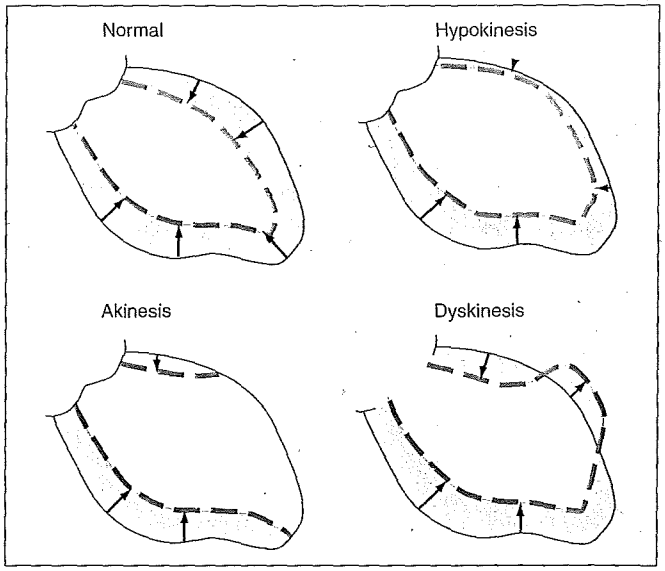

FIGURE 13-6

Diagrammatic representation of end-diastolic (*solid line*) and end-systolic (*dashed line*) silhouettes of left ventricular cineangiograms. These represent various forms of localized wall motion disorder in patients with coronary heart disease. Normal wall motion is symmetric; a patient with *hypokinesis* exhibits reduced contraction, seen here over the anterior and apical surfaces; a patient with *akinesis* exhibits absent wall motion, seen here over the anteroapical surface; a patient with *dyskinesis* exhibits paradoxic bulging of a small portion of the anterior wall with systole.

CORONARY ANGIOGRAPHY

While the aspects of cardiac catheterization described above may be performed in specific clinical situations, the essence of modern catheterization is high-quality coronary angiography. The tips of specially shaped catheters are placed into the left and then the right coronary artery, and any surgical bypass grafts, under fluoroscopic guidance. Hand injection of a radiographic contrast agent allows opacification of their lumina, with those images recorded at 15 frames per second on a radiographic image (*cine angiography*). Each coronary artery is usually viewed in several projections to permit assessment of the location and severity of any stenosis relative to the adjacent "normal" vessel segments (Fig. 13-8). In addition to the detection of coronary artery stenoses, coronary angiography evaluates the rapidity of coronary flow, the blush of capillary filling in the myocardium, collateral pathways that perfuse myocardial territories supplied by an occluded vessel, the presence of congenital abnormalities of the coronary circulation (e.g., coronary fistulae), and patency of any previously constructed coronary artery bypass grafts.

The degree of stenosis is typically evaluated by visual estimation of percent diameter stenosis of each lesion relative to the "uninvolved" adjacent reference segment, with 50% diameter stenosis representing the minimal

Q1

With progressive worsening of a proximal epicardial artery stenosis, the distal resistance vessels (when they function normally) dilate to reduce vascular resistance and maintain coronary blood flow. A pressure gradient develops across the proximal stenosis, and poststenotic pressure falls. When the resistance vessels are maximally dilated, myocardial blood flow becomes dependent on the pressure in the coronary artery distal to the obstruction. In these circumstances, ischemia, manifest clinically by angina or electrocardiographically by ST-segment deviation, can be precipitated by increases in myocardial oxygen demand caused by physical activity, emotional stress, and/or tachycardia. Changes in the caliber of the stenosed coronary artery due to physiologic vasomotion, loss of endothelial control of dilation (as occurs in diabetes mellitus), pathologic spasm (Prinzmetal's angina), or small platelet-rich plugs can also upset the critical balance between oxygen supply and demand and thereby precipitate myocardial ischemia.

EFFECTS OF ISCHEMIA

During episodes of inadequate perfusion caused by coronary atherosclerosis, myocardial tissue oxygen tension falls and may cause transient disturbances of the mechanical, biochemical, and electrical functions of the myocardium. Coronary atherosclerosis is a focal process that usually causes nonuniform ischemia. Regional disturbances of ventricular contractility cause segmental akinesia or, in severe cases, bulging (dyskinesia), which can greatly reduce myocardial pump function.

The abrupt development of severe ischemia, as occurs with total or subtotal coronary occlusion, is associated with almost instantaneous failure of normal muscle contraction and relaxation. The relatively poor perfusion of the subendocardium causes more intense ischemia of this portion of the wall (in comparison with the subepicardial region). Ischemia of large portions of the ventricle causes transient LV failure, and if the papillary muscle apparatus is involved, mitral regurgitation can occur. When ischemia is transient, it may be associated with angina pectoris; when it is prolonged, it can lead to myocardial necrosis and scarring with or without the clinical picture of acute MI (Chap. 35).

A wide range of abnormalities in cell metabolism, function, and structure underlie these mechanical disturbances during ischemia. The normal myocardium metabolizes fatty acids and glucose to carbon dioxide and water. With severe oxygen deprivation, fatty acids cannot be oxidized, and glucose is degraded to lactate; intracellular pH is reduced, as are the myocardial stores of high-energy phosphates, i.e., ATP and creatine phosphate. Impaired cell membrane function leads to the leakage of potassium and the uptake of sodium by myocytes, as well as an increase in cytosolic calcium. The severity and duration of the imbalance between myocardial oxygen supply and demand determine whether the damage is reversible (≤20 min for total occlusion in the absence of collaterals) or whether it is permanent, with subsequent myocardial necrosis (>20 min).

Ischemia also causes characteristic changes in the electrocardiogram (ECG) such as repolarization abnormalities, as evidenced by inversion of T waves and, when more severe, by displacement of ST segments (Chap. 11). Transient T-wave inversion likely reflects nontransmural, intramyocardial ischemia; transient ST-segment depression often reflects subendocardial ischemia; and ST-segment elevation is thought to be caused by more severe transmural ischemia. Another important consequence of myocardial ischemia is electrical instability, which may lead to isolated ventricular premature beats or even ventricular tachycardia or ventricular fibrillation (Chap. 16). Most patients who die suddenly from IHD do so as a result of ischemia-induced ventricular tachyarrhythmias (Chap. 29).

ASYMPTOMATIC VERSUS SYMPTOMATIC IHD

Postmortem studies on accident victims and military casualties in western countries have shown that coronary atherosclerosis often begins to develop prior to 20 years of age and is widespread even among adults who were asymptomatic during life. Exercise stress tests in asymptomatic persons may show evidence of silent myocardial ischemia, i.e., exercise-induced ECG changes not accompanied by angina pectoris; coronary angiographic studies of such persons may reveal coronary artery plaques and previously unrecognized obstructions (Chap. 13). Postmortem examination of patients with such obstructions without a history of clinical manifestations of myocardial ischemia often shows macroscopic scars secondary to MI in regions supplied by diseased coronary arteries, with or without collateral circulation. According to population studies, ~25% of patients who survive acute MI may not come to medical attention, and these patients carry the same adverse prognosis as those who present with the classic clinical picture of acute MI (Chap. 35). Sudden death may be unheralded and is a common presenting manifestation of IHD (Chap. 29).

Patients with IHD can also present with cardiomegaly and heart failure secondary to ischemic damage of the LV myocardium that may have caused no symptoms prior to the development of heart failure; this condition is referred to as *ischemic cardiomyopathy*. In contrast to the asymptomatic phase of IHD, the symptomatic phase is characterized by chest discomfort due to either angina pectoris or acute MI (Chap. 35). Having entered the symptomatic phase, the patient may exhibit a stable or progressive course, revert to the asymptomatic stage, or die suddenly.

STABLE ANGINA PECTORIS

This episodic clinical syndrome is due to transient myocardial ischemia. Males constitute ~70% of all patients with angina pectoris and an even greater fraction of those

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TABLE 17-1
ETIOLOGIES OF HEART FAILURE

Depressed Ejection Fraction (<40%)	
Coronary artery disease	Nonischemic dilated cardiomyopathy
Myocardial infarction[a]	Familial/genetic disorders
Myocardial ischemia[a]	Infiltrative disorders[a]
Chronic pressure overload	Toxic/drug-induced damage
Hypertension[a]	Metabolic disorder[a]
Obstructive valvular disease[a]	Viral
Chronic volume overload	Chagas' disease
Regurgitant valvular disease	Disorders of rate and rhythm
Intracardiac (left-to-right) shunting	Chronic bradyarrhythmias
Extracardiac shunting	Chronic tachyarrhythmias
Preserved Ejection Fraction (>40–50%)	
Pathological hypertrophy	Restrictive cardiomyopathy
Primary (hypertrophic cardiomyopathies)	Infiltrative disorders (amyloidosis, sarcoidosis)
Secondary (hypertension)	Storage diseases (hemochromatosis)
Aging	Fibrosis
	Endomyocardial disorders
Pulmonary Heart Disease	
Cor pulmonale	
Pulmonary vascular disorders	
High-Output States	
Metabolic disorders	Excessive blood-flow requirements
Thyrotoxicosis	Systemic arteriovenous shunting
Nutritional disorders (beriberi)	Chronic anemia

[a]*Note:* Indicates conditions that can also lead to heart failure with a preserved ejection fraction.

most patients with CAD. Both CAD and hypertension interact to augment the risk of HF, as does diabetes mellitus.

In 20–30% of the cases of HF with a depressed EF, the exact etiologic basis is not known. These patients are referred to as having nonischemic, dilated, or idiopathic cardiomyopathy if the cause is unknown (Chap. 21). Prior viral infection or toxin exposure (e.g., alcoholic or chemotherapeutic) may also lead to a dilated cardiomyopathy. Moreover, it is becoming increasingly clear that a large number of the cases of dilated cardiomyopathy are secondary to specific genetic defects, most notably those in the cytoskeleton. Most of the forms of familial dilated cardiomyopathy are inherited in an autosomal dominant fashion. Mutations of genes encoding cytoskeletal proteins (desmin, cardiac myosin, vinculin) and nuclear membrane proteins (lamin) have been identified thus far. Dilated cardiomyopathy is also associated with Duchenne's, Becker's, and limb girdle muscular dystrophies. Conditions that lead to a high cardiac output (e.g., arteriovenous fistula, anemia) are seldom responsible for the development of HF in a normal heart. However, in the presence of underlying structural heart disease, these conditions can lead to overt HF.

GLOBAL CONSIDERATIONS

Rheumatic heart disease remains a major cause of HF in Africa and Asia, especially in the young. Hypertension is an important cause of HF in the African and African-American populations. Chagas' disease is still a major cause of HF in South America. Not surprisingly, anemia is a frequent concomitant factor in HF in many developing nations. As developing nations undergo socioeconomic development, the epidemiology of HF is becoming similar to that of Western Europe and North America, with CAD emerging as the single most common cause of HF. Although the contribution of diabetes mellitus to HF is not well understood, diabetes accelerates atherosclerosis and is often associated with hypertension.

PROGNOSIS

Despite many recent advances in the evaluation and management of HF, the development of symptomatic HF still carries a poor prognosis. Community-based studies indicate that 30–40% of patients die within 1 year of diagnosis and 60–70% die within 5 years, mainly from worsening HF or as a sudden event (probably

Kirk, James

MRN: 000021110978

Description: 51 year old male

Regional Outreach Services
9/28/2016
BLACK RIVER FALLS KROHN CLINIC

Provider: Ward M Brown, MD (CARDIOLOGY)
Reason for visit: Referred by Deb Tidquist, NP

Progress Notes

Unsigned

James Kirk 000021110978

CARDIOLOGY BLACK RIVER FALLS
September 28, 2016

OUTREACH OUTPATIENT VISIT

Mr. Kirk is a 51-year-old white male who has a history of ischemic heart disease status post myocardial infarction and multiple angioplasties with 1 stent placed, by report; was originally seen by me back in January 27, 2016, for the evaluation and treatment of shortness of breath and postprandial abdominal discomfort. The patient was recently hospitalized at Gundersen Health for evaluation and treatment of chest pain (The patient's hospitalization was 09/06/2016 to 09/07/2016).

SUBJECTIVE:
The patient reports that since he was released from the hospital he has been experiencing no further chest discomfort. He does note his heart is "pounding " when he is recumbent at night lying on his side. He denies any orthopnea or PND and he has had no ankle swelling, save for at the end of the day when he has had some mild swelling of the right ankle. He has been taking his medications as prescribed and he offers no other complaints.

OBJECTIVE:
Vital Signs: Blood pressure in the right arm is 117/82, pulse is 85 and regular, respiratory rate 16, temperature 36.2, O2 saturation on room air is 96%.
General appearance: That of a 51-year-old white male with slightly above average height, medium build, who is well groomed, speaks in complete sentences and is in no apparent distress.
Skin: Turgor is good.
HEENT: PERRLA/EOMI. Conjunctivae and sclerae clear.
Neck: Supple.
Lungs: Clear to auscultation and percussion without rales, rhonchi, or wheezes noted.
Heart: Reveals the PMI to be in the 5th intercostal space in the midclavicular line. No heaves or thrills are palpated. There is a regular rate and rhythm. S1, S2 normal. No gallop, murmur, or rub.

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Extremities: No cyanosis or edema.

I reviewed with the patient the result of his cardiac catheterization and echocardiogram which were obtained during his hospitalization earlier this month. The patient's questions were answered and he voiced understanding.

We discussed that at this point, there is "nothing going on" heart wise that warrants anymore angioplasties or mechanical revascularization. The patient's questions were answered. He voiced understanding. We recommend that he continue with his medications as before, noting that his medications very well may need to be titrated depending on his clinical condition. Patient voiced understand.

PLAN:
1. Continue with current medications.
2. Continue with cardiac risk factor reduction efforts.
3. He will follow up with Debra L. Tidquist, nurse practitioner, as planned.
4. Follow up in this office on an as-needed basis.

Ward M Brown, MD, FACC
DD: 09/28/2016 11:42 DT: 09/29/2016 11:49 DOC#: 714941453 MODL

CC: Debra L. Tidquist, APRN-BC

Additional Documentation

Encounter Info: Billing Info, History, Allergies, Detailed Report

Orders Performed

CONSULT TO CARDIOLOGY CLINIC

Medication Changes

None

Visit Diagnoses

None

Printed by Diane C Huber at 10/3/16 6:50 AM

T2

rhythm, and it may improve the success of electrical cardioversion in patients with HF. Amiodarone increases the level of phenytoin and digoxin and prolongs the INR in patients taking warfarin. Therefore it is often necessary to reduce the dose of these drugs by as much as 50% when initiating therapy with amiodarone. The risk of adverse events, such as hyperthyroidism, hypothyroidism, pulmonary fibrosis, and hepatitis, are relatively low, particularly when lower doses of amiodarone are used (100–200 mg/d).

Implantable cardiac defibrillators (ICDs; see below) are highly effective in treating recurrences of sustained ventricular tachycardia and/or ventricular fibrillation in HF patients with recurrent arrhythmias and/or cardiac syncope, and they may be used as stand-alone therapy or in combination with amiodarone and/or a beta blocker (Chap. 16). There is no role for treating ventricular arrhythmias with an antiarrhythmic agent without an ICD.

DEVICE THERAPY

Cardiac Resynchronization Approximately one-third of patients with a depressed EF and symptomatic HF (NYHA class III–IV) manifest a QRS duration >120 ms. This ECG finding of abnormal inter- or intraventricular conduction has been used to identify patients with dyssynchronous ventricular contraction. The mechanical consequences of ventricular dyssynchrony include suboptimal ventricular filling, a reduction in LV contractility, prolonged duration (and therefore greater severity) of mitral regurgitation, and paradoxical septal wall motion. *Biventricular pacing,* also termed *cardiac resynchronization therapy* (CRT) stimulates both ventricles near simultaneously, thereby improving the coordination of ventricular contraction and reducing the severity of mitral regurgitation. When CRT is added to optimal medical therapy in patients in sinus rhythm, there is a significant decrease in patient mortality and hospitalization, a reversal of LV remodeling, as well as improved quality of life and exercise capacity. Accordingly, CRT is recommended for patients in sinus rhythm with an EF <35% and a QRS >120 ms and those who remain symptomatic (NYHA III–IV) despite optimal medical therapy. The benefits of CRT in patients with atrial fibrillation have not been established.

Implantable Cardiac Defibrillators (See also Chap. 16) The prophylactic implantation of ICDs in patients with mild to moderate HF (NYHA class II–III) has been shown to reduce the incidence of sudden cardiac death in patients with ischemic or nonischemic cardiomyopathy. Accordingly, implantation of an ICD should be considered for patients in NYHA class II–III HF with a depressed EF of <30–35% who are already on optimal background therapy, including an ACE inhibitor (or ARB), a beta blocker, and an aldosterone antagonist. An ICD may be combined with a biventricular pacemaker in appropriate patients.

MANAGEMENT OF HF WITH A PRESERVED EJECTION FRACTION (>40–50%) Despite the wealth of information with respect to the evaluation and management of HF with a depressed EF, there are no proven and/or approved pharmacologic or device therapies for the management of patients with HF and a preserved EF. Therefore, it is recommended that initial treatment efforts should be focused, wherever possible, on the underlying disease process (e.g., myocardial ischemia, hypertension) associated with HF with preserved EF. Precipitating factors, such as tachycardia or atrial fibrillation, should be treated as quickly as possible through rate control and restoration of sinus rhythm when appropriate. Dyspnea may be treated by reducing total blood volume (dietary sodium restriction and diuretics), decreasing central blood volume (nitrates), or blunting neurohormal activation with ACE inhibitors, ARBs, and/or beta blockers. Treatment with diuretics and nitrates should be initiated at low doses to avoid hypotension and fatigue.

ACUTE HF

Defining an Appropriate Therapeutic Strategy The therapeutic goals for the management of acute HF therapy are to (1) stabilize the hemodynamic derangements that provoked the symptoms responsible for the hospitalization, (2) identify and treat the reversible factors that precipitated decompensation, and (3) reestablish an effective outpatient medical regimen that will prevent disease progression and relapse. In most instances this will require hospitalization, often in an intensive care unit (ICU) setting. Every effort should be made to identify the precipitating causes, such as infection, arrhythmias, dietary indiscretion, pulmonary embolism, infective endocarditis, occult myocardial ischemia/infarction, environmental and/or emotional or environmental stress (Table 17-3), since removal of these precipitating events is critical to the success of treatment.

The two primary hemodynamic determinants of acute HF are elevated LV filling pressures and a depressed cardiac output. Frequently the depressed cardiac output is accompanied by an increase in systemic vascular resistance (SVR) as a result of excessive neurohormonal activation. Because these hemodynamic derangements may occur singly or together, patients with acute HF generally present with one of four basic hemodynamic profiles (Fig. 17-3): normal LV filling pressure with normal perfusion (Profile A), elevated LV filling pressure with normal perfusion (Profile B), elevated LV filling pressures with decreased perfusion (Profile C), and normal or low LV filling pressure with decreased tissue perfusion (Profile L).

Accordingly, the therapeutic approach to treating patients with acute HF should be tailored to reflect the patient's hemodynamic presentation. The goal should be, whenever possible, to restore the patient to a normal

:K, JAMES

ID:000021110978

07-SEP-2016 05:16:08

Gundersen Health System-3CC ROUTINE RECORD

)CT-1964 (51 yr)
.e Caucasian

)m:HM38~
:218

Vent. rate	83	BPM
PR interval	220	ms
QRS duration	154	ms
QT/QTc	400/470	ms
P-R-T axes	45 20	232

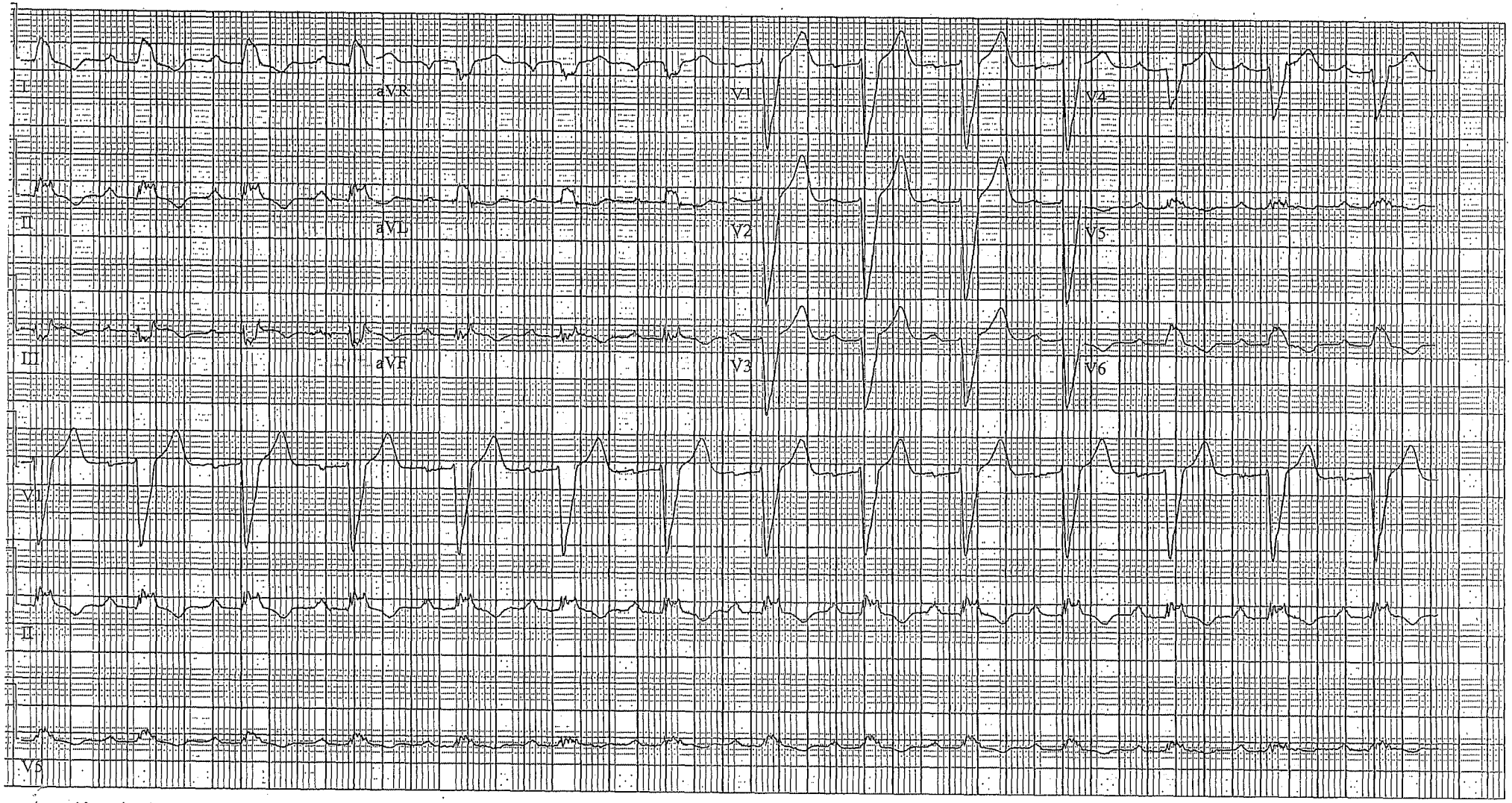
Sinus rhythm with 1st degree A-V block
QTc prolongation mild.
Left bundle branch block
Abnormal ECG
When compared with ECG of 06-SEP-2016 18:05, (unconfirmed)
No significant change was found
Confirmed by Johnson, Gordon L. (3816) on 9/7/2016 9:16:15 AM

Technician: RJN
Test ind:

MMENT:

Referred by: CHIH-SHENG CHIANG

Confirmed By: Gordon L. Johnson



mm/s 10mm/mV 150Hz 8.0 SP2 12SL 241 CID: 0

EID:3816 EDT: 09:16 07-SEP-2016 ORDER: 85872378 ACCOUNT: 117845181

NEW YORK HEART ASSOCIATION CLASSIFICATION

FUNCTIONAL CAPACITY	OBJECTIVE ASSESSMENT
Class I	Patients with cardiac disease but without resulting limitation of physical activity. Ordinary physical activity does not cause undue fatique, palpitations, dyspnea, or anginal pain.
Class II	Patients with cardiac disease resulting in slight limitation of physical activity. They are comforatable at rest. Ordinary physical activity results in fatique, palpitation, dyspnea, or anginal pain.
Class III	Patients with cardiac disease resulting in marked limitation of physical activity. They are comforatable at rest. Less than ordinary activity causes fatique, palpitation, dyspnea, or anginal pain.
Class IV	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of heart failure or the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

Source: Adapted from New York Heart Assossiation, Inc., Diseases of the Heart and Blood Vessels: Nomenclature and Criteria for Diagnosis, 6th ed. Boston, Little Brown, 1964, p. 114.

W1

DEPARTMENT OF CORRECTIONS
Division of Adult Institutions
DOC-3035 (Rev. 9/2016)

HEALTH SERVICE REQUEST AND COPAYMENT DISBURSEMENT AUTHORIZATION

WISCONSIN
Adm. Code
Ch. DOC 316

NOTIFY ANY FACILITY STAFF IF YOUR HEALTH CARE NEED IS AN EMERGENCY

PRINT LAST NAME	PRINT FIRST NAME	DOC NUMBER
Kirk	James	119291
FACILITY NAME	**HOUSING UNIT**	**TODAY'S DATE**
JCI	M 111	11-27-17

COPAYMENT DISBURSEMENT REQUEST SECTION

AGREEMENT BY PATIENT:

I understand the following:

- The Department of Corrections shall charge a copayment of $7.50 for a visit (face to face contact) initiated by a patient when a copayment is required.
- I will not be denied care if I am unable to pay the copayment.
- By signing below, I am initiating a request for disbursement of my funds for the copayment at the time of the visit when a copayment is required.
- Failure to sign below will NOT prevent the copayment from being withdrawn from my account following a visit when a copayment is required.

PATIENT SIGNATURE ▶

TO BE COMPLETED BY HSU ONLY

☐ MEDICAL (Nurse, Doctor/NP/PA) ☐ DENTAL ☐ OPTICAL

Charge Copayment: ☐ Yes ☐ No

AUTHORIZED STAFF SIGNATURE	DATE OF SERVICE

TO BE COMPLETED BY INMATE PATIENT – HEALTH SERVICE REQUEST SECTION

Be sure to include today's date on top of form. Check the appropriate box below, and explain your request on the lines provided. Place all 4 pages of the completed form in the sick call box. The HSU will send a copy back to you indicating that your request has been received.

☒ HEALTH SERVICES ☐ HEALTH CARE RECORD REVIEW ☐ COPIES FROM HEALTH CARE RECORD (List records below)
☐ PSYCHIATRIST ☐ INFORMATION
☐ OTHER:

Please provide a brief description below of the services you desire so that HSU can respond to your request appropriately.

I still have ringing in my ears, and poor circulation since the 9-6-16 Angioplasty, which is keeping my toe from healing. I still have pain near my liver when I stand for a long time, it may be because of my back.

DATE RECEIVED: TO BE STAMPED BY HSU

REC'D NOV 28 2017

FOLD THE BOTTOM OF THE FORM UP TO THE DOTTED LINE SO THAT INFORMATION REMAINS CONFIDENTIAL.

PATIENT: DO NOT WRITE BELOW THIS LINE – TO BE COMPLETED BY HSU ONLY

HSU RESPONSE Check appropriate box below. Add written comments / information as needed.

☒ Nursing Sick Call: ☒ Today ☐ Date (if not today):

☒ Scheduled to be seen in HSU ☒ ACP ☐ RN/LPN ☐ Special Needs Evaluation ☐ Optical ☐ Other:

☐ Refer HSR to: ☐ ACP ☐ HSU Manager ☐ Psychiatrist ☐ MPAA ☐ Optical ☐ Other:

☐ Refer for copies only: ☐ Refer for Health Care Record review appointment.

☐ Educational material attached (Specify): ☐ Other:

COMMENT / INFORMATION

PRINT STAFF NAME	DATE OF HSU RESPONSE
Kla Bubera RN	11-28-17

ORIGINAL - PATIENT REQUEST FOLDER

X1

DEPARTMENT OF CORRECTIONS
Division of Adult Institutions
DOC-3035 (Rev. 9/2016)

HEALTH SERVICE REQUEST AND COPAYMENT DISBURSEMENT AUTHORIZATION

WISCONSIN
Adm. Code
Ch. DOC 316

NOTIFY ANY FACILITY STAFF IF YOUR HEALTH CARE NEED IS AN EMERGENCY

PRINT LAST NAME	PRINT FIRST NAME	DOC NUMBER
Kirk	James	119291
FACILITY NAME	**HOUSING UNIT**	**TODAY'S DATE**
JCI	M	12-10-17

COPAYMENT DISBURSEMENT REQUEST SECTION

AGREEMENT BY PATIENT:

I understand the following:

- The Department of Corrections shall charge a copayment of $7.50 for a visit (face to face contact) initiated by a patient when a copayment is required.
- I will not be denied care if I am unable to pay the copayment.
- By signing below, I am initiating a request for disbursement of my funds for the copayment at the time of the visit when a copayment is required.
- Failure to sign below will NOT prevent the copayment from being withdrawn from my account following a visit when a copayment is required.

PATIENT SIGNATURE ▶

TO BE COMPLETED BY HSU ONLY

☐ MEDICAL (Nurse, Doctor/NP/PA) ☐ DENTAL ☐ OPTICAL

Charge Copayment: ☐ Yes ☐ No

AUTHORIZED STAFF SIGNATURE	DATE OF SERVICE

TO BE COMPLETED BY INMATE PATIENT - HEALTH SERVICE REQUEST SECTION

Be sure to include today's date on top of form. Check the appropriate box below, and explain your request on the lines provided. Place all 4 pages of the completed form in the sick call box. The HSU will send a copy back to you indicating that your request has been received.

☒ HEALTH SERVICES ☐ HEALTH CARE RECORD REVIEW ☐ COPIES FROM HEALTH CARE RECORD (List records below)
☐ PSYCHIATRIST ☐ INFORMATION
☐ OTHER:

Please provide a brief description below of the services you desire so that HSU can respond to your request appropriately.

My hearts been hurting the last couple of days, I still have ringing in my ears, And poor circulation I don't know if the infection in my toes is going to my heart or what, but its been hurting.

DATE RECEIVED: TO BE STAMPED BY HSU

RECD DEC 11 2017

FOLD THE BOTTOM OF THE FORM UP TO THE DOTTED LINE SO THAT INFORMATION REMAINS CONFIDENTIAL.

PATIENT: DO NOT WRITE BELOW THIS LINE – TO BE COMPLETED BY HSU ONLY

HSU RESPONSE Check appropriate box below. Add written comments / information as needed.

☒ Nursing Sick Call: ☒ Today ☐ Date (if not today):

☒ Scheduled to be seen in HSU ☒ ACP ☐ RN/LPN ☐ Special Needs Evaluation ☐ Optical ☐ Other:

☐ Refer HSR to: ☐ ACP ☐ HSU Manager ☐ Psychiatrist ☐ MPAA ☐ Optical ☐ Other:

☐ Refer for copies only: ☐ Refer for Health Care Record review appointment.

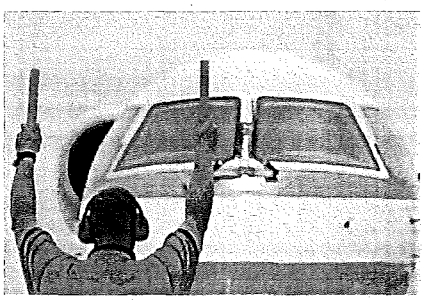
☐ Educational material attached (Specify): ☐ Other:

COMMENT / INFORMATION

PRINT STAFF NAME	DATE OF HSU RESPONSE
Kristine Pralle RN	12/11/17

ORIGINAL - PATIENT REQUEST FOLDER

Y1



SLIDESHOW

Top Caus of Seve Hear Loss

Start >

Some instances of tinnitus are caused by infections or blockages in the ear, and the tinnitus can disappear once the underlying cause is treated. Frequently, however, tinnitus continues after the underlying condition is treated. In such a case, other therapies -- both conventional and alternative -- may bring significant relief by either decreasing or covering up the unwanted sound.

What Causes Tinnitus?

Prolonged exposure to loud sounds is the most common cause of tinnitus. Up to 90% of people with tinnitus have some level of noise-induced hearing loss . The noise causes permanent damage to the sound-sensitive cells of the cochlea, a spiral-shaped organ in the inner ear. Carpenters, pilots, rock musicians, street-repair workers, and landscapers are among those whose jobs put them at risk, as are people who work with chain saws, guns, or other loud devices or who repeatedly listen to loud music. A single exposure to a sudden extremely loud noise can also cause tinnitus.

A variety of other conditions and illnesses can lead to tinnitus, including:

- Blockages of the ear due to a buildup of

Z 1

wax, an ear infection, or rarely, a benign tumor of the nerve that allows us to hear (auditory nerve)

- Certain drugs -- most notably aspirin, several types of antibiotics, anti-inflammatories, sedatives, and antidepressants, as well as quinine medications; tinnitus is cited as a potential side effect for about 200 prescription and nonprescription drugs.
- The natural aging process, which can cause deterioration of the cochlea or other parts of the ear
- Meniere's disease, which affects the inner part of the ear
- Otosclerosis, a disease that results in stiffening of the small bones in the middle ear
- Other medical conditions such as high blood pressure, cardiovascular disease, circulatory problems, anemia, allergies, an underactive thyroid gland, autoimmune disease, and diabetes
- Neck or jaw problems, such as temporomandibular joint (TMJ) syndrome
- Injuries to the head and neck

Tinnitus can worsen in some people if they drink alcohol, smoke cigarettes, drink caffeinated beverages, or eat certain foods. For reasons not yet entirely clear to researchers, stress and fatigue seem to worsen tinnitus.

WebMD Medical Reference | Reviewed by Neha Pathak, MD on February 15, 2017

Sources

NEXT ARTICLE
Symptoms >

Z 2

DEPARTMENT OF CORRECTIONS
Division of Adult Institutions
DOC-3021

WISCONSIN

PROGRESS NOTES

PATIENT NAME: KIRK, James 119291
DOB: [redacted]1964

DOC NUMBER

DATE	TIME	PROGRESS NOTES – SUBJECT, OBJECTIVE, ASSESSMENT, PLAN
12/12/17	0825	237.0 121/78 97.0 98 20 95%
		vitals —— [illegible], cont
		Cellulitis (L) 3rd toe – No
		drainage noted. also red around
		great toe – Will put on course
		of Clindamycin – No drain need
		for removal at this time.
		(signature)
12/21/17	10:45	S/O: I/M patient scheduled for follow up on
		left 3Rd toe cellulitis. Toe not warm to touch,
		no pain/tenderness to touch, 0 exudate/drainage,
		tip of toe to ~~medial~~ lateral side toe dark
		pink. Pt. states that "it feels so much better".
		A: Alt. Skin integrity R/T cellulitis (L) 3Rd
		toe P: Reinforced completion of Clindamycin
		Pt. verb. understanding. Writer informed to
		contact HSU prn next 1-2 weeks if no improvement
		or toe worsens —— Cecilia Hutcheson Smith

DISTRIBUTION: Original – Medical Chart, Progress Notes Section

AA1

DEPARTMENT OF CORRECTIONS
Office of the Secretary
DOC-400 (Rev. 1/2016)

Dr. Martin not addressing chest pain, ringing in my ears, poor circulation, and pain near my liver / back pain

WISCONSIN
Administrative Code
Chapter DOC 303.32 & 310

INMATE COMPLAINT

31978

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DATE RECEIVED	COMPLAINT CODE	COMPLAINT FILE NUMBER

INSTRUCTIONS FOR INMATE: COMPLETE ALL SECTIONS OF FORM. Do not use a highlighter or marker on this form. The form may be returned to you if you submit an incomplete form or if you do not follow the instructions. Print clearly, illegible forms will not be processed. See reverse side for more information.

INMATE NAME (if group complaint, enter name of spokesperson)	DOC NUMBER	HOUSING UNIT	FACILITY
James Kirk	119291	M	JCI

LOCATION OF INCIDENT	DATE OF INCIDENT	TIME OF INCIDENT
HSU	12-12-17	AM

ANSWER THE FOLLOWING QUESTIONS IN THE SPACES PROVIDED:

BRIEFLY STATE WHO OR WHAT IS THE **ONE** ISSUE OF THIS COMPLAINT? Deliberate indifference to my serious medical needs.

PRIOR TO SUBMITTING THIS COMPLAINT, HOW DID YOU ATTEMPT TO RESOLVE YOUR **ONE** ISSUE AND WITH WHOM?

I wrote Ms. Tammi Maassen.

WHAT WAS THE RESULT OF YOUR ATTEMPT TO RESOLVE THE **ONE** ISSUE?

Nothing.

WHAT ARE THE DETAILS SURROUNDING THIS COMPLAINT? On 11-27-17, I put in a HSU Request saying "I still have ringing in my ears, And poor circulation since the 9-6-16 Angioplasty, which is keeping my toe from healing. I still have pain near my liver when I stand for a long time, it may be because of my back." I was seen by Nurse LaBarbera On 11-28-17, where she scheduled me to see the doctor (Dr. W Brad Martin).

On 12-10-17, I put in Another HSU Request, saying "My hearts been hurting the last couple of

SIGNATURE OF INMATE: James Kirk

DATE SIGNED: 12-16-17

Continue on reverse if more space is needed.

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BB1

days, I still have ringing in my ears, and poor circulation, I don't know if the infection in my toes is going to my heart or what, but its been hurting." Nurse Kristine Pralle called me up to HSU on 12-11-17 to ascertain that I was not having a heart attack or it was not an emergency situation. She told me I had an Appt. with Dr. Martin on the next day, 12-12-17. We both agreed that I would be OK till then.

At my Appt. with Dr. Martin on 12-12-17, he prescribed [redacted] Clindamycin 300 mg 3 times a day for 10 days. He felt the pulse in my foot to confirm I have poor circulation. I told him I have been having bad ringing in my ears, and poor circulation since the

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BB2

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Office of the Secretary
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INMATE COMPLAINT

31978

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James Kirk	119291	M	JCI
LOCATION OF INCIDENT	**DATE OF INCIDENT**	**TIME OF INCIDENT**	
HSU	12-12-17	AM	

ANSWER THE FOLLOWING QUESTIONS IN THE SPACES PROVIDED:

BRIEFLY STATE WHO OR WHAT IS THE **ONE** ISSUE OF THIS COMPLAINT? See page 1

PRIOR TO SUBMITTING THIS COMPLAINT, HOW DID YOU ATTEMPT TO RESOLVE YOUR **ONE** ISSUE AND WITH WHOM?

See page 1

WHAT WAS THE RESULT OF YOUR ATTEMPT TO RESOLVE THE **ONE** ISSUE?

See page 1

WHAT ARE THE DETAILS SURROUNDING THIS COMPLAINT? Angioplasty. My limbs keep getting numb and going to "sleep." He said "the Angioplasty would not cause this." I said "I get some papers on Tinnitis and it can be caused by poor circulation or cardiovascular disease." He said "the tinnitis could be caused by anything." I said "I got it right after the Angioplasty." I then asked him "if we could get an echocardiogram and a stress test to figure it out, also the numbers on the previous echocardiograms don't match," he said "We

SIGNATURE OF INMATE

DATE SIGNED 12-16-17

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BB3

don't need it." He never listened to my heart, never asked me about the chest pain, did nothing about the Tinnitus or poor circulation. I said "I still have pain near my liver," he said "it could be caused by my back." He did absolutely nothing about this either. The pain is excruciating when I stand for 10 or 15 minutes, or sit in a padded chair. It radiates from my spine, around my torso, painful nerve pain. He totally ignored every issue but my toe.

ACTION REQUESTED Take me to a doctor to address my medical needs for starters, And he needs to find out the true nature of the damage to my heart. You can't fix it unless you know what is actually wrong with it.

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BBH

ICE REPORT
COMPLAINT NUMBER JCI-2017-31978
* * * ICRS CONFIDENTIAL * * *

To: KIRK, JAMES L. - #119291
UNIT: _MB1 -- _111_L
JACKSON CORRECTIONAL INSTITUTION
PO Box 233
BLACK RIVER FALLS, WI 54615-0233

Complaint Information:

Date Complaint Acknowledged: 12/18/2017 Inmate Contacted? No

Date Complaint Received: 12/18/2017

Subject of Complaint: 4 - Medical

Brief Summary: Inmate claims his medical needs are not being addressed. He requests seeing a doctor who,'needs to find out the true nature of the damage to my heart.'

Summary of Facts: Inmate Kirk complains when he met with Dr. Martin, he did nothing about his concerns regarding the ringing in his ears and poor circulation, which he contends he has had since his angioplasty in 2016. He also complained of pain near his liver. He asked Dr. Martin to order an angiogram, and was told "we don't need it." He was also told the pain he was having could be related to his back problems.

According to the above staff and the medical record, Inmate Kirk was seen by Dr. Martin on 12/12/17. At that visit, Dr. Martin addressed concerns with his toenail infection and ordered Clindamycin. Dr. Martin did not order anything further regarding his other complaints.

Inmate Kirk has made it clear he is not satisfied with the care being offered to date, but what type of specific care or treatment must be offered is a matter of professional medical judgment. Documentation indicates he continues to be seen by medical staff for his medical problems and there is no indication the treatment offered is not adequate to the demonstrated need. As this complaint was reviewed by the HSU Manager, no further action is recommended.

ICE Recommendation: Dismissed

Recommendation Date: 01/09/2018

J. Dougherty

J. Dougherty - Institution Complaint Examiner

Nurse Cecilia Smith called me up to check on my toes like Dr. Martin ordered on 12-12-17. She did not take my vitals.

DEPARTMENT OF CORRECTIONS
Office of the Secretary
DOC-400 (Rev. 1/2016)

WISCONSIN
Administrative Code
Chapter DOC 303.32 & 310

INMATE COMPLAINT

569

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James Kirk	119291	M	JCI

LOCATION OF INCIDENT	DATE OF INCIDENT	TIME OF INCIDENT
HSU	12-21-17	AM

ANSWER THE FOLLOWING QUESTIONS IN THE SPACES PROVIDED:

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PRIOR TO SUBMITTING THIS COMPLAINT, HOW DID YOU ATTEMPT TO RESOLVE YOUR **ONE** ISSUE AND WITH WHOM?

Yes, Tammi Maassen.

WHAT WAS THE RESULT OF YOUR ATTEMPT TO RESOLVE THE **ONE** ISSUE?

Nothing.

WHAT ARE THE DETAILS SURROUNDING THIS COMPLAINT? On 12-21-17, Nurse C. Smith called me up to HSU to look at my infected toes. On 12-12-17, Dr. Martin prescribed Clindamycin 300 mg. 3x daily for 10 days. She did not take my vitals. When I seen Dr. Martin, I told him about my poor circulation (that is part of the reason my toes are healing slowly), And how My Ankles were hurting And my feet And hands go to sleep, And how my ears ring, And the pain near my liver was not going away. Nurse Smith looked at my toes, I told her that it

SIGNATURE OF INMATE

DATE SIGNED
1-4-18

Continue on reverse if more space is needed.

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DD1

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WISCONSIN
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Chapter DOC 303.32 & 310

was doing a little better. I told her that the Clindamycin was making my circulation way worse, my ankles were hurting worse, my feet and hands were going to sleep, and the ringing in my ears was way worse. It felt like I was allergic to the Antibiotic. She just said keep taking them. I told her I need a foot tub and some Epsom salts, she said they didn't do that, but they made an exception. She said she would write and let me know. She did not. She did Absolutely Nothing at this visit when I told her the Clindamycin was bad for me.

ACTION REQUESTED Send me to a competent doctor that can give me an ECG and a stress test, find out why my circulation is so poor, quit concealing the true nature of my medical condition. I'm still feeling bad from that Clindamycin.

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INMATE COMPLAINT

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INMATE NAME (if group complaint, enter name of spokesperson)	DOC NUMBER	HOUSING UNIT	FACILITY
James Kirk	119291	M	JCI
LOCATION OF INCIDENT	**DATE OF INCIDENT**	**TIME OF INCIDENT**	
HSU	12-21-17	AM	

ANSWER THE FOLLOWING QUESTIONS IN THE SPACES PROVIDED:

BRIEFLY STATE WHO OR WHAT IS THE **ONE** ISSUE OF THIS COMPLAINT? Attachment to JCI-2018-569.

PRIOR TO SUBMITTING THIS COMPLAINT, HOW DID YOU ATTEMPT TO RESOLVE YOUR **ONE** ISSUE AND WITH WHOM?

—

WHAT WAS THE RESULT OF YOUR ATTEMPT TO RESOLVE THE **ONE** ISSUE?

—

WHAT ARE THE DETAILS SURROUNDING THIS COMPLAINT? In the original complaint, I forgot to list that my chest pain was a lot worse. I was basically having an allergic reaction to the Clindamycin, and she said to keep taking it, and she did not even take my vitals. I don't know if I had a bad reaction because I am allergic to Tetnus vaccine, DTP, or what, but it was not good.

Thank you.

SIGNATURE OF INMATE

DATE SIGNED 1-6-18

Continue on reverse if more space is needed.

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ACTION REQUESTED

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Clindamycin (cont.)

Medical and Pharmacy Editor: John P. Cunha, DO, FACOEP

What Are Warnings and Precautions for Clindamycin?

Warnings

Clostridium difficile-associated diarrhea (CDAD) has been reported and may range in severity from mild diarrhea to fatal colitis.

C difficile produces toxins A and B, which contribute to CDAD; hypertoxin-producing *C difficile* strains increase morbidity and mortality (more likely to be refractory to antimicrobial therapy and may require colectomy).

If CDAD suspected or confirmed, ongoing antibiotic use not directed against *C difficile* may need to be discontinued.

This medication contains clindamycin. Do not take Cleocin, Cleocin Pediatric, Clindesse, or ClindaMax Vaginal if you are allergic to clindamycin or any ingredients contained in this drug.

Keep out of reach of children. In case of overdose, get medical help or contact a Poison Control Center immediately.

Contraindications

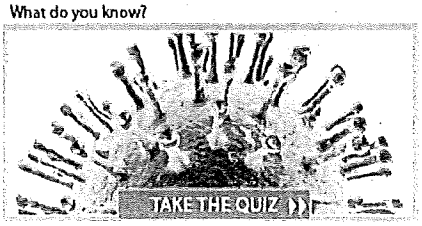
Hypersensitivity to clindamycin, lincomycin, or formulation components

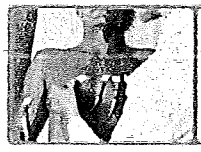
Effects of Drug Abuse

None.

Short-Term Effects

See "What Are Side Effects Associated with Using Clindamycin?"





EE 1

Long-Term Effects

Risk of potentially fatal pseudomembranous colitis, fungal or bacterial superinfection on prolonged use; discontinue therapy if significant abdominal cramps, diarrhea, or passage of blood and mucus occurs.

See "What Are Side Effects Associated with Using Clindamycin?"

Cautions

Endocarditis prophylaxis: Use only for high-risk patients, per recent AHA guidelines.

Risk of potentially fatal pseudomembranous colitis, fungal or bacterial superinfection on prolonged use; discontinue therapy if significant abdominal cramps, diarrhea, or passage of blood and mucus occurs.

May increase risk of drug-resistant bacteria if prescribed in the absence of proven or strongly suspected bacterial infection.

Use caution in hepatic impairment, monitor for hepatic abnormalities; periodic liver enzyme determinations should be made when treating patients with severe liver disease.

Not for use in meningitis due to inadequate penetration into CSF.

Severe skin reactions including toxic epidermal necrolysis, drug reaction with eosinophilia and systemic symptoms (DRESS), and Stevens-Johnson syndrome (SJS), some with fatal outcome, reported; permanently discontinue if reactions occur.

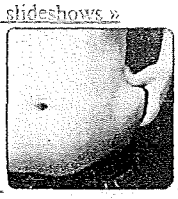
Parenteral product contains benzyl alcohol, which has been associated with gasping syndrome and death in newborns.

Use with caution in patients with history of gastrointestinal disease, especially colitis.

Not for administration as a bolus; infuse over 10-60 minutes.

Consider possibility of clostridium difficile in all patients who present with diarrhea following antibiotic use.

Serious anaphylactic reactions requiring immediate emergency treatment with



Prescribe with caution in atopic individuals.

Indicated surgical procedures should be performed in conjunction with antibiotic therapy.

Clindamycin dosage modification may not be necessary in patients with renal disease.

Discontinue therapy permanently and institute appropriate therapy if anaphylactic or severe hypersensitivity reaction occurs.

Pregnancy and Lactation

Clindamycin may be acceptable for use during pregnancy. Either animal studies show no risk but human studies not available or animal studies showed minor risks and human studies done and showed no risk.

In clinical trials with pregnant women, the systemic administration of clindamycin during the second and third trimesters, has not been associated with an increased frequency of congenital abnormalities

Clindamycin is excreted in breast milk; the manufacturer suggests discontinuing the drug or do not nurse (AAP Committee states compatible with nursing).

Medical Editor: John P. Cunha, DO, FACOEP

SOURCE:

Medscape. Clindamycin.

EE2

Clindamycin

Medical and Pharmacy Editor: John P. Cunha, DO, FACOEP

Brand Name(s): Cleocin, Cleocin Pediatric, Clindesse, ClindaMax Vaginal

Generic Name: clindamycin

Drug Class: Antibiotics, Lincosamide

What Is Clindamycin and How Does It Work?

Clindamycin is indicated in the treatment of serious infections caused by susceptible anaerobic bacteria.

Clindamycin is also indicated in the treatment of serious infections due to susceptible strains of streptococci, pneumococci, and staphylococci. Its use should be reserved for penicillin-allergic patients or other patients for whom, in the judgment of the physician, a penicillin is inappropriate. Because of the risk of colitis, before selecting clindamycin, the physician should consider the nature of the infection and the suitability of less toxic alternatives (e.g., erythromycin).

Clindamycin is available under the following different brand names: Cleocin, Cleocin Pediatric, Clindesse, and ClindaMax Vaginal.

Dosages of Clindamycin

Adult and Pediatric Dosage Forms & Strengths

Capsule

- 75 mg

EE3

Kirk, James

November 3, 2017

Subjective: Patient presents for a routine followup hepatitis C treatment now after week 24 visit. No complaining of flu like symptoms or fatigue. Appetite is stable. No episodes of jaundice. Tolerated treatment with Epclusa well without side effects

Objective: Vital signs: General: No apparent distress. Skin is warm and dry with no jaundice or pruritus. HEENT is within normal limits. Neck is supple no lymphadenopathy or thyromegaly. Lungs are clear bilaterally to auscultation and percussion of the colonic distention heart is normal S1 and S2 no murmurs rubs or gallops. Abdomen is soft and nontender no ascites. No spider hemangiomatas. No hepatosplenomegaly or masses are felt. Lower extremities are free from edema pedal pulses are equal bilaterally at 2+. No clonus.

Patient was extensively counseled on avoidance of acetaminophen containing products as well as other liver toxic medications. He is also counseled him as to avoid sharing needles, razors, tooth brushes or nail clipper to prevent the spread of hepatitis C to others. Patient is also counseled that if he would become in contact with contaminated blood products that he could potentially reobtain hepatitis C

Labs: are as reviewed and discussed with patient hepatitis C quantitative viral load is not detected.

Assessment: Hepatitis C genotype 3 sustained responder

Plan: Patient was counseled as above. We will discontinue medical hold as well as hepatitis C chronic disease group.

Debora Tidquist FNP-BC

Debra Tidquist FNP BC

DEPARTMENT OF CORRECTIONS
Division of Adult Institutions
DOC-3021 (Rev. 8/2007)

WISCONSIN

PROGRESS NOTES

PATIENT NAME Last	First	DOC NUMBER
Kirk, James	DOC# 119291	DOB '54

DATE	TIME	PROGRESS NOTES – SUBJECT, OBJECTIVE, ASSESSMENT, PLAN
7-28-16	@1240	Inmate returned from offsite orders and instructions were reviewed Will follow up with provider as indicated Offsite forwarded to provider for review —— Debra
8/10/16	1410	228 97.7 120/86 85 97% T. Helgr

Kirk, James DOC# 119291

August 10, 2016

Subjective: Patient is seen to follow up on spirometry test. States he still becomes short of breath with activity. Denies orthopnea. Able to sleep flat with one pillow. He states he is also having epigastric type pain. Despite being on ranitidine 150 mg twice a day. Denies chest pain, sweating profusely or back in neck or jaw.

Objective: Weight is 228 temp 97.7 blood pressure 120/86 pulse 85 sats are 97% on room air. Reviewed previous cardiology notes again with patient, as well as spirometry results with patient. The patient completed a pre/ post bronchodilator spirometry which showed an FVC of 4.71 which is 85% of predicted the FEV1 was 4.86 which is a 1% decline in the patient's FEV1 was 3.20 which is a 77% of predicted with a post bronchodilator dilator and a FEV1 of 3.13 which is a 2% decline. FEV1 to FVC ratio was 68% the patient's flow-volume loop was most consistent with obstructive pulmonary deficits. On exam patient appears to be in no acute distress. He is able to converse in complete sentences without becoming short of breath. Neck is supple lungs are clear bilaterally to auscultation and percussion with slightly prolonged expiratory phase. No barrel chest. Heart is normal S1 and S2 no murmurs rubs or gallops. Abdomen is soft and non-tender no hepatosplenomegaly or masses are felt. Bowel sounds are present in all 4 quadrants.

Assessment: #1. Mild obstructive pulmonary disease. #2. Coronary artery disease status post myocardial infarction with multiple angioplasties is in one stent placed. #3. Hypertension is in good control with current medications. #4. Dyslipidemia. #5 Epigastric pain not relieved with ranitidine.

Plan: Counseled on COPD and progression of disease, medications used to treat. Also counseled patient extensively not to restart smoking to prevent further damage to lungs. Hand out on COPD was given. We will start on albuterol inhaler 2 puffs 4 times a day when necessary for one year will follow-up in one month. RN is to instruct him on proper use of inhaler. We will draw an H. pylori antibody to see if this is the cause of his epigastric complaints. We will also consider possible stress sestamibi and evaluation and follow-up with cardiologist. We will continue to work to maximize his carvedilol dose as well as his ACE inhibitor dose. We have started him diuretic per cardiology recommendations at previous visits.

Debra Tidquist FNP-BC

Debra Tidquist FNP-BC

8/16/16	0730	H. Pylori Ab ⊖ —— Debra Tidquist FNP-BC

DISTRIBUTION: Original – Medical Chart, Progress Notes Section

GG1

* Auth (Verified) *

KIRK, JAMES
DOB: 10/24/1984
DOS: 05/08/2017
Attn: Tidquist, Debra
FIN: 445125
MRN: 202200

Gundersen Health System
La Crosse, WI 54601
Transthoracic Echocardiographic Report
Study Date: 5/8/2017

ICAEL

Name: James Kirk MRN: 021110978
Provider: DEB TIDQUIST NP DOB: 1964 Age: 52 years
Indication: ABDOMINAL PAIN

Conclusions:
1. Normal LV ejection fraction, calculated at 68%.
2. Mild concentric left ventricular hypertrophy.
3. Prior examinations are available and were reviewed for comparison purposes.
4. This is an abnormal echocardiogram with detailed findings discussed below.

Description of Findings:
Study: 2D, M-mode, color Doppler and spectral Doppler. The study quality was technically limited, due to imaging difficulties.
Cardiac Rhythm: Is not able to determine rhythm.
Left Ventricle: The left ventricular internal cavity size was normal. Normal segmental wall motion. Mild concentric left ventricular hypertrophy. Spectral Doppler shows insufficient data to estimate pattern of LV diastolic filling. False tendon in left ventricular apex.
Left Atrium: Intra-atrial septum is intact. Left atrial size is normal.
Right Ventricle: Normal right ventricular size and function.
Right Atrium: The right atrium is normal. Right atrial pressure is estimated to be normal.
Aortic Valve: The aortic valve is tricuspid, mildly sclerotic and leaflet mobility is non restricted. No indication of aortic valve regurgitation is seen.
Mitral Valve: The mitral valve is degenerative. Mitral leaflet mobility is normal. No evidence of mitral valve stenosis. Mild mitral valve regurgitation is seen.
Tricuspid Valve: The tricuspid valve is normal in appearance. No tricuspid regurgitation is visualized.
Pulmonic Valve: The pulmonic valve is normal in structure. No indication of pulmonic valve regurgitation.
Pulmonary Artery: The pulmonary artery is of normal size and origin.
Aorta: The aortic arch is normal at 3.0 cm. The ascending aorta is normal and measures 3.05 cm in diastole.
Venous: Inferior vena cava is normal with normal inspiratory collapse.
Pericardium: No pericardial effusion is seen.

All Measurements and Calculations on next page.

6/6/17

Final

Facility: BRM Hospital

REC'D JUN 0 2 2017

HH1

* Auth (Verified) *

James Kirk 021110978 5/8/2017

2-D and M-Mode Measurements:

	3D	M-Mode	NL Values Men	NL Value Female		Calculations
LVIDd	5.0 cm		4.2-5.5	3.8-5.2	LV EF% (Apex)	
LVIDs	4.3 cm				HR	
IVSd	1.3 cm		0.6-1.0	0.6-0.9	BSA	63.48 m²
LVPWd	1.0 cm		0.6-1.0	0.6-0.9	LV Mass (I BSA)	
Ao Asc(d)	3.0 cm		<3.7 cm			
LA (s)			2.0-4.0 cm		Ao Asc Zscore	
RV			<3.7 cm			
LA (Vol)	0.9 ml/m²		<20 cm			
RA (area)						

Diastology:

Mitral		NL Values	Pulmonary	NL Values	Hemodyn		NL Values
E Peak:	1.0 m/s		Pulm S		Prop Vel		>50cm/s
A Peak:	0.4 m/s		Pulm D		E'	0.10 m/s	
E/A	2.4	1.1-1.5	D dt		A'	m/s	
DT		160-240 ms	ar Vel	<30cm/s	E/E'	10	<12
A dur			ar Dur				
IVRT		50-90msec					

Aortic:

		NL Values			Severe		Severe
Vmax	1.0 m/s		AVA		<.8	AI slope	
TVI	0.15 m		Max G	4.1 mmHg	>64mmHg	AI P 1/2	<500 ms
LVOT V	0.8 m/s		Mean G	2.6 mmHg	>40mmHg	AI ERO	
LVOT TVI	0.12 m		DOI	0.76	<.25	AI Vol	
LVOT dia			Planim AV		<.8	AI RF%	
						CEQ Vol	

Mitral:

	Severe		Severe
MVA	<1.0cm2	MR ERO	>40mm2
P 1/2t	>180ms	MR Vol	>60ml
Mean G	>10mmHg	MR radius	
MV TVI		CEQ Vol	
MV plan		MR TVI	

Tricuspid:

		NL Values		NL Values		NL Values
TR Vmax			TR ERO	>40mm2	PV Vmax 0.8 m/s	
TR maxG			TR Vol	>30ml	PV meanG 1.5	
RAP		>5 mmHg			PADP	
RVSP		>35mmHg				
IVC insp	cm					
IVC exp	cm	<2.2 cm				

Pulmonic:

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HH2

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James Kirk 021110978 5/8/2017

Height:	182.90 cm	HR:	bpm
Weight:	103 kg	BP:	/
BSA:	63.48 m²	Gender:	M

Sonographer: KAD
Site: BRFMH
Location:OUTREACH
Imaging Limitations:
Contrast/IV documentation:

KIRK, JAMES
DOB: 10/24/1964
DOS: 05/08/2017
Attn: Tidquist, Debra
FIN: 445125
MRN: 202200

Electronically Signed By: 1001101 Esosa Odigie-Okon MD
5/8/2017, 2:58:14 PM

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